A STUDY OF THE EFFECTIVENESS OF COGNITIVE THERAPY AND PHARMACOTHERAPY, EACH ALONE, AND IN COMBINATION IN THE TREATMENT OF DEPRESSED OUTPATIENTS

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SUMMARY OF THE THESIS

The three main aims of this research were: (1) to compare a specific and time-limited form of psychotherapy, namely cognitive therapy, with pharmacotherapy (drug of choice) in the treatment of depression, (2) to test out the effect of combining cognitive therapy and pharmacotherapy (drug of choice) and (3) to investigate the efficacy of these modes of treatment relative to one another in two populations, outpatients from a teaching hospital and from the community, i.e. a general practice.

Depressed patients were referred from two sources, a hospital outpatient clinic and a general practice clinic, and were screened using a standard psychiatric interview, the Present State Examination, for elicitation of symptoms and signs. On the basis of this interview, Spitzer's research diagnostic criteria were checked. All of the patients admitted to the study satisfied the criteria for primary major depressive disorder. In addition, level of self-reported depression had to be at least mild according to the British norms of the Beck Depression Inventory (i.e. $\geq$14).

Eighty-eight patients were randomly assigned to pharmacotherapy (drug of choice), individual treatment with cognitive therapy or a combination of pharmacotherapy (drug of choice) and individual cognitive therapy. For the cognitive therapy and combination treatment, the protocol specified a maximum of 20 weeks, with both cognitive therapy groups attending twice a week at the beginning and once a week thereafter. The pharmacotherapy patients were prescribed therapeutic doses appropriate to each drug. If no response occurred (i.e. at least a 50% reduction in level of depression) after a maximum of twelve weeks of treatment, patients were dropped from the trial whatever type of treatment they received.

In the hospital sample all three treatment groups showed statistically significant decreases in depressive symptoms. Combination treatment resulted in greater improvement than did cognitive therapy alone and pharmacotherapy alone on a number of mood and cognitive measures.

In the general practice population, combination treatment and cognitive therapy alone resulted in statistically significant decreases in depressive symptoms whereas pharmacotherapy, on the whole, failed to reduce depressive symptoms. Both combination treatment and cognitive therapy alone resulted in significantly greater decreases in depressive symptoms than did pharmacotherapy on several measures of mood and a cognitive measure.

When responders only were examined, pharmacotherapy, cognitive therapy and combination treatment produced different patterns of response over the treatment period in terms of self-reported depression and hopelessness. Combination treatment in particular, but cognitive therapy as well, resulted in a quicker and sharper effect than pharmacotherapy, especially on hopelessness, a variable that is reported to correlate highly with suicidal intent.

In terms of attrition, no one treatment was better than any other in reducing the number of drop outs from therapy in each population.

It was concluded that cognitive therapy is at least as effective as pharmacotherapy in the treatment of hospital outpatients and that it is more effective than pharmacotherapy in depressed general practice patients. Further, the combination of pharmacotherapy and individual cognitive therapy may be particularly useful in the treatment of chronically depressed hospital outpatients.

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This thesis has been composed by myself. The work reported is part of a project in which other colleagues were also involved. Dr. Blackburn, the other clinical psychologist involved in this research, collected data from thirty-three hospital outpatients and has kindly permitted these data to be included in the analysis. My contribution to this work involved

selecting, and collecting data from, all of the general practice patients as well as sixteen hospital outpatients. In addition, the author made a substantial contribution to the planning of the methodology and all of the statistical analysis was computed by myself. My own contribution to this work is further indicated in the text.

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CONTENTS

CHAPTER ONE

INTRODUCTION

A. GENERAL AIM OF THE RESEARCH

This thesis is directed towards examining the scope and limitations of cognitive-behavioural therapy, a theoretically-based, pragmatic and time-limited psychological approach to the treatment of depression. The decision to investigate the efficacy of this treatment, relative to traditional pharmacological methods, was considerably influenced by the sequence of research studies which have been undertaken by A.T. Beck and his associates at the University of Pennsylvania and by other researchers who, over the last twenty years, have investigated the role of thinking in the development of human emotions and behaviours.

B. DEFINITION OF DEPRESSION

Depression is one of the oldest recognised disorders of psychological life. The Biblical figure Job suffered from severe depression and feelings of self-reproach; in the first century Plutarch graphically described behaviours characteristic of the disturbance; in 1621 Sir Robert Burton devoted an entire treatise to 'The Anatomy of Melancholy' (Zilboorg and Henry, 1949), and in the twentieth century depression is said to have become so widespread that it has been referred to as 'the common cold of mental illness' (Miller and Seligman, 1973, P.62). Some authors have been quick to attribute its prevalence to technological, social and economic pressures; others have been more reserved in their search for explanations. Whatever its ultimate

cause, one thing is clear: the management of clinical depression ranks as one of the most challenging tasks in modern psychiatry.

Depression is typically thought of as a mood or affective disorder. Some opening remarks concerning affect-related terms may be useful here. Nowlis (1963) regards affects as hierarchically ordered phenomena; in order of increasing degree the sequence is composed of emotions, moods and temperaments. Higher degrees of affect last longer than lower ones. In combination, higher degree affects afford the background activity with which environmental stimuli interact in the manifestation of affective responses. Emotional states relate to situation-specific affects, whereas emotional traits refer to a predisposition to experience certain emotional states with consistent and somewhat predictable latencies, intensities, and durations (Spielberger, 1972); there is substantial overlap between the concepts of mood and affective trait. According to some theorists, then, depression may be conceptualised as an affective trait, predisposition, or mood (Becker, 1977).

Although a large literature exists on state and trait anxiety as a personality construct, relatively little has been written about state and trait depression. Behaviourally oriented theorists have shown a tendency to apply the concept of anxiety to all dysphoric states and temperaments but, as will become clear, this situation has changed considerably in recent years.

As Becker (1974, 1977) notes, moods are not entirely affective occurrences; they are associated with thoughts and overt behaviours. Cognitively oriented researchers regard moods as a class of responses to an intervening evaluation of antecedent events. This evaluation may well entail a cognitive bias (Beck, 1967; Lazarus, 1968). Such a position underscores the importance of thinking style in the aetiology

of depression and forms the theoretical backbone of this thesis. Some authors focus on the motivational characteristics of affects (Brown and Farber, 1951; Tomkins, 1962); others concentrate on their operant properties (Ferster, 1974; Lewinsohn, 1974). Some of these different approaches and their implications for depression as a set of clinical problems will be discussed shortly.

The label 'depression' may refer to a state of 'feeling low', or a simple case of 'the blues', to a symptom, a syndrome, or a disease (Mendels, 1968). As Greenacre (1953) says: 'Depression as a symptom is as ubiquitous as life itself and, in a mild degree, appears 'naturally' as a reaction to loss which need hardly be questioned'. When, then, is depression a malady? She concludes, 'it is certainly the intensity, the excessive duration, and the domination of the organism by the affect, rather than its occurrence which is pathological' (P.9).

Although the difference between a mild bout of 'feeling low' and depression is, in many ways, a matter of degree, within the field of psychiatry depression is also regarded as a qualitative deviation from the norm. Because there are no signs or symptoms that are unique to depression, it is frequently associated with many other psychopathological conditions and some physical illnesses (Blumenthal, 1971). Not surprisingly, the study and treatment of clinical depression is plagued by the problem of low diagnostic reliability (Zubin, 1967; Kendell, 1975).

Burns and Beck (1978) have summarised the descriptive aspects of depression:

> 'Depression is a disorder of the entire psychobiologic system including the emotions, thoughts, behaviours and somatic functions. The emotional component is characterised by a blue mood involving feelings of sadness, anhedonia, guilt, irritability and despair.

> The somatic functions include hypochondriasis, insomnia, or hypersomnia, weight gain or loss, constipation or diarrhoea, fatigue and decreased libido. The behavioural changes are characterized by passivity, lethargy, inactivity, social isolation, withdrawal from work and avoidance of pleasurable activities. In some patients there is an associated anxiety component that consists of fear, apprehension, and a sense of impending doom. Frank panic attacks may be accompanied by somatic sensations such as a jumpy stomach, tingling fingers, rapid breathing, and lightheadedness.' (P. 109)

As noted, none of these symptoms invariably accompanies depression and none is wholly specific to depression.

C. THE MAGNITUDE OF THE PROBLEM

Although the study of depression is fraught with disagreement and semantic, diagnostic and aetiological problems, the fact remains that depression occupies a prominent place in the scientific investigation of mental disturbance. Depression certainly seems to account for a relatively large proportion of in-patient and out-patient cases in psychiatric hospitals. For example, the 1978 admission figures for the Royal Edinburgh Hospital show that, of the 2,401 admissions, 319 or 13.3 per cent were given a diagnosis of depression according to the International Classification of Diseases Code (1965). Moreover, the admission figures for 1978 obtained from the Scottish Home and Health Department show that, of the 26,207 admissions to all Scottish psychiatric hospitals, 13.3 per cent of the 14,305 women

admitted and 6.9 per cent of the 11,902 men admitted had been classified as suffering from a depression.* Although twice as many women had received a diagnosis of depression, the single most common diagnosis given to males was alcoholism, or alcoholic psychosis (approximately 33 per cent). With regard to alcoholism, it has been suggested that alcohol abuse may be an attempt by some individuals, particularly males, to control depression and anxiety (Goss and Morasko, 1970). Thus, the excessive use of alcohol may mask depression in some people.

Further evidence of the size of the problem which depression presents to the field of mental health can be seen in Brown and Harris's (1978) study of the prevalence of depression in Camberwell, a large, predominently working-class Inner London borough. They found that as many as 8 per cent of women surveyed at random had been psychiatrically disturbed during the year preceding contact with a trained interviewer. Although these community cases were, on the whole, less severely disturbed than an identified group of psychiatric patients, those sampled did seek medical attention at some point during their depressive episode.

Recently, Weissman and Myers (1978) investigated the prevalence of affective disorders in a United States urban community using an interview schedule based on Spitzer's Research Diagnostic Criteria (1978). The main finding was that primary major depression was the most common disorder in the community with a current prevalence rate of 4.3 per cent.

The range of findings for population studies varies

* The figures have been calculated using four diagnostic categories: 296.1 (manic-depressive psychosis, depressed type), 296.3 (manic-depressive psychosis, circular type but currently depressed), 298.0 (depressive type), and 300.4 (neurotic depression).

considerably. Many of these variations may be explained by variations in methodology, especially in the diagnostic criteria used. It is likely that this accounts for discrepancies in community survey data and, as will become clear, admission rates between the United Kingdom and America. In general, however, epidemiological studies have estimated that from four to twenty-four per cent of the population may experience episodes of depression severe enough to warrant clinical intervention (Schwab et al., 1968). Depression self-report symptom scales (e.g. Zung, Beck Depression Inventory) when applied to community samples account for the relatively higher estimates (Weissman et al., 1978).

There is also evidence that, in populations of medically ill patients, depression is frequently underdiagnosed and is inappropriately treated even when recognised (Davidson et al., 1973; Kotin et al., 1973; Lehmann, 1968; Raft, 1975). In a community survey, for example, Craig et al. (1978) examined the relationship between current drug use and symptoms of depression. Forty-two per cent of 771 men and sixty per cent of 1,059 women reported having used one or more medications in the 48 hours prior to an interview. The use of medications and the number of medications used increased progressively with age among both men and women. Respondents who used four or more drugs included significantly more high scorers on a depression checklist than those who used fewer medications. Moreover, the group of women who used minor tranquilizers and sedatives included significantly more high depression scorers than those who did not. In both men and women, those scoring in the depressed range who were getting psychotropic drugs tended to be taking minor tranquilizers or sedatives. The authors concluded:

> 'At the present time, if depression is recognized at all, it is most likely to be treated with non-specific remedies (minor tranquilizers and sedatives) that, at best, offer merely palliative symptomatic relief and, at worst, may intensify the distress they are intended to treat.' (P.1039)

Similarly, diazepam and other antianxiety agents, not antidepressants, were the drugs most commonly prescribed for depressed individuals in a sample of middle class people seeking marital and sexual counseling (Gullick and King, 1979); in a door to door survey of symptoms present and drugs used (Uhlenhuth et al., 1978); and in family practice patients (Hesbacher et al., 1976). Thus, depression might be one psychiatric syndrome for which diazepam and similar drugs are most often inappropriately prescribed.

Inappropriate treatment (antianxiety agents) of depression is a matter for concern because the disturbance may result in suicide. There appear to be many other aspects of the morbidity, as well as the mortality, of depression that warrant appropriate treatment. For instance, Weissman and Klerman (1977) in an investigation of chronic depression found the use of antianxiety medication (rather than antidepressants) to be one of the three most important factors correlated with chronicity of depression, the others being evidence of neurotic personality patterns predating the acute episode, and not having received maintenance treatment beyond the acute phase.

According to the National Institute of Mental Health, depression accounts for seventy-five per cent of all psychiatric hospitalizations in the United States and during any given year fifteen per cent of all adults between the ages of 18 and 74 may experience significant depressive symptoms (Secunda, 1973, in Beck, 1978). Secunda also reports

that twenty-five per cent of hospitalised depressed patients and ten per cent of diagnosed depressed patients are psychotic by rigorous criteria, i.e. diagnosed on the basis of socio-environmental criteria and the presence of biological signs of depression rather than on the basis of a thought disorder such as delusions, hallucinations, or impaired reality testing (in Becker, 1977). It must be noted that these diagnostic criteria are not generally accepted in the United Kingdom where delusions and hallucinations are considered as the sine qua non of a psychotic illness (Wing et al., 1974; Foulds, 1976). Certainly, the different emphasis on symptoms in the two countries helps to explain the considerably lower admission figures found in Scottish psychiatric hospitals.

As suggested earlier, for some people a depressive episode ends fatally. Stengel (1970) has reported that the number of people attempting suicide in a large metrapolitan city such as London with a population of eight million is between 7500 and 12000 per year; for England and Wales the figure is between 30000 and 40000 per year. The estimated mean annual suicide rate per 100000 population in England and Wales for the period 1965-1973 inclusive is reported to be 10.75 for men and 7.4 for women (Myers and Neal, 1978). With regard to suicide attempts, many are not successful and many are not the result of depression. Nevertheless, it is generally recognised that the most vulnerable people to death by suicide are those who suffer from depression. For example, retrospective studies (Robins et al., 1959; Dorpot and Ripley, 1960; Flood and Seager, 1968; Barraclough et al., 1974) have shown that in 80 to 90 per cent or more of deaths from suicide, the victim had experienced psychiatric disturbance at the time of death, most frequently depression, but often

alcoholism.

Secunda (1973, in Becker, 1977) reported that in the United States the suicide rate for the general population is 0.01 per cent, while it is about fourteen per cent for individuals suffering from bipolar affective disorder, i.e. a history of depression and mania. The author also points out that approximately 23,000 people commit suicide in the United States per year, this in spite of advances in the chemotherapy of depression. Miles (1977) collected evidence from a variety of sources and concluded that almost all of the suicides in America can be attributed to depressive illness, followed closely by alcoholism, then schizophrenia, neurosis and personality disorder, and drug addiction.

In his paper 'Suicide in Britain', Brown (1979) states that, while doctors have an important potential role in preventing suicide among depressed patients, this potential has yet to be realised and that primary prevention may not have had much impact on suicidal behaviour. For instance, he cites the study by Barraclough et al. (1974) in which 93 of 100 consecutive suicides had visited a doctor within one year of death, and 48 within one week of death. Of these 24 had sought help from a psychiatrist. In another study Murphy (1975, also in Brown) found that of 49 suicides who had presented to a doctor within six months of dying, 35 had threatened or tried to commit suicide, but only 39% of the doctors were aware of this history. Similarly, Myers and Neal (1978) reported that very few of the psychiatric patients in their investigation were receiving adequate physical treatment for depression at the time of death, yet 63 per cent had consulted a physician within a month beforehand. Thus, it seems there is room for improvement with respect to the prevention

of suicide. As Brown (1979) recommends, it may be that more effective education of physicians, including psychiatrists, in the diagnosis and application of physical treatments of depression is required in order to achieve this goal. However, a potentially useful alternative to drug treatment for the depressed, suicide-prone individual might be the kind of psychological treatment being investigated in this thiesis, namely cognitive-behavioural therapy.

What is the likelihood that an individual will develop a depressive illness in his lifetime? Secunda (1973, in Becker, 1977) estimated that the lifetime probability of developing a severe depressive episode is from four to ten per cent, though a higher figure (18 per cent) is reported by Weissman et al. (1978). Consistent with hospital admission figures, the prevalence rate for women is usually reported as being twice that of men (Weissman and Klerman, 1977). Apart from the anguish which depression causes for the individual and his family, particularly in view of the recurrent nature of the illness, the treatment of depressive disorders places considerable strain on the resources of any health service.

Given the relatively high prevalence, morbidity, and mortality of depression, it is clear that depression must be regarded as a major health problem. Physicians find it hard to diagnose depression, especially at the primary care level. Even if it is recognised in the community, it is likely to be treated with non-specific remedies which may simply perpetuate an already debilitating illness. Techniques for the prevention of suicidal behaviour (which is on the increase in Britain and the United States (Brown, 1979)) are in need of improvement in both the primary care and psychiatric clinic. Finally, the probability of an individual developing a depressive

disorder during his lifetime, particularly women, is quite high.

D. THE NEED FOR AN ALTERNATIVE TREATMENT

What kind of interventions are needed to deal with these problems? As will be discussed in the next chapter, the appropriate use of physical methods of treatment (ECT, tricyclics, MAOIs and lithium) has provided a partial answer to this question. For example, there is strong evidence that physical treatments, in particular ECT and tricyclic drugs, work very well for some depressed patients. However, various investigations and reviews (e.g. MRC Trial, 1965; Quitkin et al., 1976; Morris and Beck, 1974) indicate that a significant percentage of patients do not recover at all, and for those who do respond, recovery may be slow and incomplete with frequent relapses.

In light of the deficiencies in the therapeutic response of depressed patients, it would appear to be worthwhile to investigate alternative methods of treatment for depression, in particular those psychological techniques which have received the most persuasive empirical support. Recently, cognitive and behavioural psychologists and psychiatrists have shown an interest in affective disorders which has led to the development of a set of psychological techniques for the treatment of depressions (Mahoney, 1974; Seligman, 1975; Lewinsohn, 1974; Ferster, 1974; Rehm, 1977; Beck, 1967, 1973, 1976, 1978, 1979). Several factors would appear to account for this heightened interest:

(1) the less than satisfactory results obtained by physical methods of treatment as outlined above

(2) evidence for the efficacy of behavioural, or performance-based therapies for the treatment of problems such as phobias,

test anxiety, stuttering, bedwetting, etc.

(3) the growing awareness that a pluralistic approach is perhaps the most reasonable stance to take with respect to the aetiology of depression (Akiskal and McKinney, 1975)

(4) the increasing amount of empirical support for the role of cognition within the freamework of social learning theory (Bandura, 1977) to explain human behaviour in general,

and specifically,

(5) the growing awareness that thinking style may play an important role in the development and maintenance of emotional disorders.

E. FORMAT OF THE REVIEW

The following chapters will take up these points in more detail. Before that, a review of physical treatments of depression is presented, highlighting deficiencies which, in spite of the not inconsiderable success of this therapeutic approach, undermine the adequacy of a purely biological model of depressive illness. Chapter two will also consider the evidence for the use of drugs in combination with psychotherapy. The third chapter will consider the impact which the various psychosocial theories, particularly behavioural approaches, have made on the study of depression, as well as their therapeutic implications. Chapter four, the final review section, will examine the theory and supporting evidence which underlies the research reported in this thesis, that is, the cognitive approach to the treatment of clinical depression.

CHAPTER TWO

DRUGS, ELECTROCONVULSIVE THERAPY AND THE USE OF DRUGS IN COMBINATION WITH PSYCHOTHERAPY IN THE TREATMENT OF DEPRESSION

A. PHARMACOLOGICAL TREATMENTS

The affective disorders vary in severity from mild but nevertheless distressing self-limiting alterations in mood, many of which never come to medical attention, right through to the most severe and disabling psychotic states. For clinical depression, urgent measures are indicated for psychotic symptoms, depressive stupor and suicidal intent. The depressive syndrome also leads to considerable morbidity with impairment of domestic, social and occupational aspects of life. However, with regard to treatment, the tricyclic compounds and monoamine oxidase inhibitors introduced over twenty years ago have been reported to provide symptom relief in a significant proportion of depressed patients (Davis et al., 1968; Smith et al., 1969; Morris and Beck, 1974; Rogers and Clay, 1975; WHO, 1975). Other drugs reported to have antidepressant effects include the tetracyclic compounds, lithium, iprindole, some phenothiazines and diazepam (Rush, 1975).

This section of the review presents a selective overview of research findings on the efficacy of pharmacological treatment in depression, focusing primarily on outcome studies in which tricyclics, monoamine oxidase inhibitors and the relatively new tetracyclic compounds are used. As a cornerstone for the chapter, the author has relied heavily on several key

reviews (e.g. MRC Trial, 1965; Morris and Beck, 1974; Paykel and Coppen, 1979) because they represent the most comprehensive and critical summary to date on the current status of antidepressant medication. In addition, a search through Psychological Abstracts was made in order to survey drug studies conducted between January 1, 1973 and March 31, 1979. It was found that many articles reported investigations on new drugs, particularly tricyclic derivatives, many of which are not marketed in the United Kingdom, findings on groups containing very few subjects, i.e. less than fifteen in each cell, and studies in which experimental drugs not in widespread use clinically (e.g. neurotransmitter precursor substances such as L-tryptophan) were tested and shown to produce equivalent effects on depressive symptoms relative to tricyclic control groups. These investigations are not included in the review. Also excluded were studies of inpatient populations in which it was clear that schizophrenics with 'secondary depressive symptoms' had been included in the sample (e.g. Raskin et al., 1975).

Original articles which report findings on relatively large populations in which the more effective antidepressants are used are selectively summarised. Moreover, the survey includes primarily those investigations that meet the following criteria: (a) the use of a control group, (b) random assignment of patients to treatment groups, (c) double-blind research design, (d) relatively large sample sizes, i.e. greater than 30 subjects. Thus, an attempt is made to overcome weaknesses of previous studies (as discussed in Beck, 1967, 1973) by defining specific inclusion criteria for the studies reviewed. As Morris and Beck (1974) point out, 'a summary of studies that meet these methodological criteria is more likely to permit a valid conclusion' (P. 667).

A survey of the recent literature revealed articles reporting studies of two or more drugs at the same time. The list of treatments reflect the separate comparisons in these investigations. Also, throughout the review the term 'treatment group' is used to indicate that the reference is to only one section of a reported study.

The subjects in the most recent survey (January, 1973 to March, 1979) had manifest clinical depression, ranged in age from twenty-one to sixty-five years and spent an average of 5.2 weeks in drug treatment. While only those investigations that met the methodological criteria outlined above are included, there was wide variation among the studies with regard to such critical variables as (1) patient characteristics, (2) length of time between treatment and evaluation, (3) method of assessment of improvement, (4) type of control condition, (5) schedule of pharmacotherapy administration, (6) physiological variations within and among patients and (7) statistical analyses. Some of the confounding effects of these variables will be taken up later.

1. Tricyclics

The tricyclic antidepressants include imipramine hydrochloride (Tofranil, Presamine), desipramine hydrochloride (Norpramin, Pertofrane), amitriptyline hydrochloride (Elavil, Tryptizol), nortriptyline hydrochloride (Aventyl), protriptyline hydrochloride (Vivactil) and doxepin hydrochloride (Sinequan). The author also found numerous reports in the recent literature which described studies on the efficacy of a new tricyclic called Amoxapine and these are also included in the review. Studies have investigated the efficacy of one or more of these drugs compared with a placebo, or the differential effects of two tricyclics and/or one of the relatively new tetracyclic compounds.

2. Tricyclics versus placebo

Morris and Beck (1974) provide an extensive review of published research which included 146 double blind studies on drugs actively promoted in the United States as antidepressants during 1972. The data indicate that, in total, tricyclics were more effective than placebo in 61 of 93 treatment groups which included inpatient, outpatient and mixed in and outpatient samples.

Rogers and Clay (1975) did a statistical review of controlled trials of imipramine and placebo in the treatment of depressive illness by extracting the basic data and analysing these by Fisher's two-tailed test. They tabulated the results of thirty studies and divided these into three diagnostic groups - endogenous, mixed endogenous/neurotic, and neurotically depressed patients. They found that in the endogenous depressed category five out of fourteen studies showed imipramine to be more effective than placebo, the drug being particularly effective for patients in an acute phase of the illness. Thirteen studies included mixed endogenous/neurotic patient samples and of these three showed imipramine to be superior to placebo, while two out of three investigations indicated the superiority of imipramine over placebo in the neurotically depressed group. In total, imipramine was more effective than placebo in ten out of thirty studies reviewed but only one of these demonstrated the efficacy of imipramine in a sample of chronically depressed patients (Leyberg et al., 1959).

As the list of studies in Table $2A_1$ indicates, the author found six comparisons of tricyclics with placebo in four investigations between 1973 and 1979. There were no studies on inpatient and mixed inpatient and outpatient samples. The four separate studies on outpatients reported significant results in favour of tricyclic drugs in all six comparisons.

Table $2A_1$ Controlled studies comparing tricyclics with placebo (1973 to 1979)

(6 tricyclic comparisons in 4 studies)

Study	Drug comparison	Type of subjects (all outpatients)	Outcome
Rickels, 1974	amitriptyline vs placebo	mixed depressed/ anxious (N = 108)	drug superior to placebo $p = < .05$
Smith, 1975	amoxapine vs imipramine vs placebo	depressed (N = 90)	both drugs superior to placebo $p = < .05$
Fabre et al., 1977	amoxapine vs imipramine vs placebo	depressed (N = 85)	both drugs superior to placebo $p = < .05$
Claghorn, 1977	imipramine vs maprotiline (tetracyclic) vs placebo	depressed (N = 45)	imipramine superior to placebo $p = < .05$

Thus, the evidence presented in this review indicates that, although equivocal results have been reported, tricyclics are more effective than placebo in the treatment of depression and the most definitive results are obtained in the treatment of non-chronic populations.

3. Imipramine and amitriptyline versus other tricyclics

Morris and Beck (1974) also reviewed studies that compared the effects of imipramine and another tricyclic drug. The data, condensed in table $2A_2$, indicates that by 1972 there were a total of thirty treatment group comparisons reported in twenty-nine studies. Four comparisons showed superiority of imipramine over other tricyclics, six treatment groups indicated that imipramine was less effective than other tricyclics, while twenty comparisons showed no significant difference between imipramine and other tricyclics.

Table $2A_2$ Number of drug group comparisons showing imipramine superior to, inferior to, or equivalent to other tricyclics

(30 treatment group comparisons in 29 studies)

Comparison drugs: desipramine, amitriptyline, nortriptyline, protriptyline, doxepin, perphenazine

Imipramine

Superior	Inferior	Equivalent effect
4	6	20

(superior and inferior indicate significant difference at $p = < .05$)

Similarly, Morris and Beck (1974) reviewed studies which compared the effects of amitriptyline and other tricyclic drugs (table $2A_3$). These indicate that in thirty-eight treatment groups in thirty-three studies, nine comparisons showed superiority of amitriptyline over other drugs, six treatment groups showed amitriptyline to be less effective than other tricyclics and twenty three groups reported no significant difference between amitriptyline and other tricyclics.

Table $2A_3$ Number of drug group comparisons showing amitriptyline superior to, inferior to, or equivalent to other tricyclics

(38 treatment group comparisons in 33 studies)

Comparison drugs: imipramine, nortriptyline, protriptyline, doxepin, perphenazine

Amitriptyline

Superior	Inferior	Equivalent effect
9	6	23

(superior and inferior indicate significant difference at $p = < .05$)

The author found four studies between 1973 and 1979 that reported on the efficacy of imipramine compared with other tricyclics and four investigations which compared amitriptyline with alternative tricyclics. As the data in table $2A_4$ indicate, imipramine was found to be superior to the little used tricyclic dimathracine when administered to a small sample of depressed inpatients (Abuzzahab, 1973). Two studies reported no significant differences between imipramine and other tricyclics (amoxapine, amitriptyline) in larger outpatient samples, though in both instances there was significant symptomatic improvement. Similarly, Smith (1975) reported an equivalent effect on symptoms for imipramine and amoxapine.

Table $2A_4$ Controlled studies comparing imipramine and amitriptyline with other tricyclics (1973 to 1979)

(8 tricyclic comparisons)

Study	Drug comparison	Type of subject	Outcome
Abuzzahab, 1973	imipramine vs dimathracine	depressed inpatients (N = 30)	imipramine superior to dimathracine $p = < .05$
Rickels, 1974	Amitriptyline vs clomipramine	depressed outpatients (N = 168)	amitriptyline superior to clomipramine $p = < .05$
Olgiati et al., 1974	amitriptyline vs pizotyline	depressed outpatients (N = 73)	equivalent effect
Smith, 1975	amoxapine vs imipramine vs placebo	depressed outpatients (N = 90)	equivalent effect generally but differential effect on symptoms
Fabre et al., 1977	amoxapine vs imipramine vs placebo	depressed outpatients (N = 85)	equivalent effect
Barnes, 1977	amitriptyline vs desipramine	depressed outpatients (N = 53)	desipramine superior to amitriptyline $p = < .05$
Yamhuire et al., 1977	amitriptyline vs amoxapine	depressed outpatients (N = 53)	equivalent effect
Goldberg et al., 1977	imipramine vs amitriptyline	depressed outpatients (N = 57)	equivalent effect

There were four investigations that compared the effects of amitriptyline with alternative tricyclics. Rickels et al. (1974) reported results on a large sample of 93 psychiatric and 75 nonpsychiatric outpatients who received either amitriptyline or clomipramine in a six week trial. The main finding was that both agents produced a high level of clinical improvement. According to several endpoint measures, amitriptyline was found to be superior to clomipramine, though there were differential effects on symptoms in the different outpatient samples.

While Morris and Beck (1974) did not report on any studies comparing amitriptyline with desipramine, the author located one study that examined the efficacy of these drugs. Barnes (1977) found desipramine to be more effective than amitriptyline ($p = < .05$) with regard to symptomatic improvement and faster onset of therapeutic action.

Finally, two studies compared the effects of amitriptyline with two of the recently investigated tricyclic compounds (Olgiati et al., 1974; Yamhure et al., 1977). In both instances no significant differences were found between amitriptyline and the experimental drugs (amoxapine, pizotyline).

In summary, the review indicates that when the effects of the conventional and most popular tricyclic antidepressants (imipramine and amitriptyline) are compared with one another and/or tricyclic derivatives, the majority of treatment comparisons (fourty-eight out of seventy-six) show no significant differences between the drugs.

4. Other comparisons and tricyclic compounds

Morris and Beck (1974) reviewed five studies in which tricyclic derivatives were compared with each other. None of the studies reported significant differences between these chemical agents. Recently, Poldinger

et al. (1978) reported an investigation on the comparative efficacy of nomifenisine and nortriptyline in a sample of depressed outpatients. Although both groups improved significantly from baseline measures, no significant difference was found between the two drugs.

The efficacy of the antidepressant iprindole, a drug closely related to doxepine, was found to be superior to placebo in three studies reviewed by Morris and Beck (1974). Iprindole has also been compared recently with imipramine in a study which involved one hundred depressed/anxious, general practice outpatients (Rickels et al., 1973). Again no significant difference was found in the effects of the two drugs, although both groups gained a high level of symptomatic improvement.

5. Monoamine oxidase inhibitors

The monoamine oxidase inhibitors (MAOI) constitute a second major class of antidepressants. Phenelzine sulfate (Nardil) and tranylcypromine sulfate (Parnate) are the two primary drugs in this category.

In 1965, an important clinical trial compared ECT, imipramine, phenelzine and placebo in the treatment of depression. Phenelzine was found to be only as effective as placebo in men and gave even less favourable results than placebo in females (MRC Trial, 1965). Morris and Beck (1974) examined eighteen studies involving tranylcypramine or phenelzine compared with imipramine and with placebo. They concluded that MAO inhibitors generally failed to show the same degree of superiority over placebo as tricyclics.

Tyrer (1976) reviewed twelve controlled trials comparing phenelzine and placebo and found that a more favourable outcome in six of the studies seemed to be associated with a larger dose than normal (45 mg/day) and continuation of treatment for six weeks.

Patients considered suitable for treatment with MAO inhibitors have been described as having 'atypical', 'reactive', or 'neurotic' depressive illness (Sargent, 1961) whereas tricyclics are said to be the drugs of choice for patients with 'typical' endogenous depressive symptomatology. Whether 'atypical depression' constitutes a valid diagnostic entity is still a matter of rigorous debate (Kendell, 1976). MAOI's have also been shown to be of benefit in anxiety states and phobic anxiety, especially agoraphobias and social phobias (Kelly, 1973, in Barnes et al., 1977).

Though earlier comparative studies, including the MRC Trial, could be criticised for inadequate dosage and duration of MAOI treatment, it would seem that in the treatment of severe depression tricyclic antidepressants are to be preferred.

6. The tetracyclics

Maprotiline

A number of investigators have compared the effects of maprotiline in depressed patients with the effects of imipramine or amitriptyline. The drug first came to medical attention in 1972 following the appearance of reports on its pharmacological and clinical characteristics. The medication is extensively reviewed by Pinder et al. (1977).

Pinto et al. (1972) reviewed thirteen double-blind trials in which maprotiline was administered in a dosage of 150 mg daily and compared with conventional antidepressants in a similar dosage. Taken together, these investigations involved 850 patients. After approximately 28 days of treatment the evaluations showed no significant differences between the efficacy of the treatments on depressive symptoms as measured by the Hamilton rating scale for depression. There was marked improvement in over 70 per cent of patients in all of the drug

conditions.

The effects of maprotiline have been investigated in a few placebo controlled studies. For example, maprotiline or placebo was administered to a matched group of nineteen patients. Equivalent effects were demonstrated between the treatment groups after one, two and four weeks of treatment. The results of this particular study are difficult to interpret because the treatment groups were ill-matched. Two thirds of the placebo group were hospital inpatients as compared with one third of the maprotiline group which also contained a large proportion of patients with a history of previous or recurrent depressive episodes (Jukes, 1975a, in Mindham, 1979). Other studies (McCallum and Meares, 1975) have reported results comparing maprotiline, amitriptyline and placebo which are equally difficult to interpret.

Some researchers have found maprotiline to have a more rapid onset of action. For instance, Pinto et al.,(op cit.) found maprotiline to be superior to imipramine in retarded depressed patients after one week's treatment, and also to amitriptyline after two weeks' administration in agitated depressed patients, but these differences were not maintained after four weeks' treatment.

Thus, the findings of investigations in which standard antidepressants were used as controls and studies which involved placebo controls are puzzling and difficult to interpret. The consensus of opinion appears to be that maprotiline may only be as effective as standard drug treatments for depression. Further studies are required to clarify the important findings reported to date.

Mianserin

The pharmacological properties of mianserin have been extensively investigated since the early 1970's while reports of its clinical efficacy have appeared since 1973.

As with maprotiline, controlled clinical trials have compared the drug with amitriptyline and imipramine. The reports indicate little difference between the drugs in terms of antidepressant effect. Some of the studies noted fewer complaints of side effects from the patients receiving mianserin (Coppen and Ghose, 1976; Coppen et al., 1976; Wheatley, 1976; Ghose et al., 1976, in Mindham, 1979).

Mindham (1979) mentions an important trial conducted by Murphy et al. (1976) in general practice because it is one of the few that involved comparison with placebo. A double-blind comparison of the effects of mianserin, imipramine and placebo was made. Imipramine and mianserin showed a similar degree of improvement and were significantly superior to placebo. The drugs were reportedly effective against the same range of symptoms and the benefit over placebo became evident after two to three weeks of treatment.

Thus, mianserin also seems to be an effective drug in the treatment of depression, though it would not appear to offer greater therapeutic advantage than the standard antidepressants.

7. Lithium carbonate

Lithium carbonate is an alkali metal which is thought to modify the distribution of electrolytes at a subcellular level (e.g. Prien et al., 1974) thereby maintaining cell electrolyte balance. Lithium has been used primarily in the management of manic-depressive illness in the manic phase, the prophylaxis of recurrent mania, and bipolar illness (Morris and Beck, 1974; Paykel and Rowan, 1979).

With regard to the prophylaxis of depression, a number of investigators have found lithium superior to placebo in the treatment of bipolar patients (e.g. Baastrup et al., 1970; Prien et al., 1973; Fieve et al., 1976). There are a few studies which indicate that lithium is more effective than placebo in unipolar patients (e.g. Coppen et al., 1971; Fieve et al., 1976). In their brief review, Paykel and Rowan (1979) note that there are no negative studies concerning the efficacy of lithium relative to placebo in the maintenance treatment of these disorders; that in bipolar depressed patients lithium was superior to imipramine; and that imipramine was 'probably' more effective than lithium in unipolar depressed patients.

Recently Worral et al. (1979) reported two randomised double-blind controlled trials on sixty-three depressed female inpatients subject to recurrent affective disorder, i.e. bipolar and unipolar psychoses. The results indicated that at the end of three weeks lithium produced greater improvement than imipramine; lithium in combination with tryptophan was more effective than tryptophan alone - the latter drug having no discernible antidepressant effect in this group of patients. The authors point out that the antidepressant activity of lithium was not apparent until the second and third week of the trial.

Hard evidence is scarce that lithium is an effective antidepressant in people who have developed a depressive disorder. Mendels (1976) has observed that an antidepressant response appears more frequently in bipolars than unipolar depressives, though in one study (Watanabe et al., 1975, in Paykel and Rowan, 1979) no difference was found between unipolars and bipolars in response to lithium which appeared to be as effective as imipramine.

Overall, there seems to be a superior maintenance effect for lithium in bipolar patients and evidence is accumulating that this preventative

effect may apply to unipolar depressives. More controlled trials should be conducted to establish further the prophylactic and possible antidepressant effects of lithium.

B. ELECTROCONVULSIVE THERAPY

The 1930's witnessed the introduction of several controversial physical therapies in the field of psychiatry (e.g. insulin shock, sleep therapy, lobotomy and electroconvulsive therapy). Of these, only ECT retains its place in the treatment of depression. ECT has been criticised on the grounds that it provides little or no therapeutic gain, that inherent dangers significantly detract from whatever benefits it might have, and still others contend that ECT should not be used because little is known about its mode of action. Freeman (1979) has pointed out that much of the criticism has been ill informed and that a considerable amount of research has examined the efficacy of ECT. This section presents a brief review of the current clinical status of ECT.

Wechsler et al. (1965) summarised research published between 1958 and 1963 which compared the effectiveness of ECT with antidepressant drug regimens. American, British and Canadian journals were the principal sources for the 153 investigations involving nearly six thousand patients. Many of the studies were uncontrolled and used extremely heterogenous patient samples. Nevertheless, the authors suggest that the 72 per cent mean improvement rate for ECT over tricyclics (65 per cent), MAOIs (50 percent) and placebo (23 per cent) gives a moderately accurate profile.

Crowe and Johnstone (1979) note that this picture probably exaggerates the efficacy of the antidepressant treatments, including ECT, because of the fact that a high proportion of these investigations were uncontrolled. Also, they demonstrated higher improvement rates than studies which include a control group.

Crow and Johnstone (1979) report a very important comparison by Wechsler et al. (1965) between investigations that deal primarily with depressions of recent onset and those which include mainly chronic depressions. Here again, the heterogeneity of the data is apparent as the studies involved some schizophrenics and geriatric patients. Table $2B_1$ extracted from their review, shows that the superiority of both drugs and ECT over placebo is much less in the category of chronic depression.

Table $2B_1$

	Mean % improvement	
	Depressions of recent onset	Chronic depressions
Total drugs	61.7	31.9
Placebo	23.7	20.7
ECT	86.1	36.7

(Crow and Johnstone, 1979, p. 109)

Two large multicentre controlled trials were published in the mid-1960s. In the United States, Greenblatt et al. (1964) reported a trial on depressed patients admitted to three state hospitals in Boston. ECT was found to be significantly more effective than all the other treatments after eight weeks (see table $2B_2$).

Table $2B_2$ A comparison of ECT with drug treatment (from Greenblatt et al., in Freeman, 1979)

Type of treatment	Results (marked improvement) %
Modified ECT (nine or more treatments)	76
Phenelzine	50
Imipramine	49
Placebo	46
Isocarboxazid	28

The Medical Research Council trial (1965) was undertaken at several centres in England. This second investigation was better designed than the previous study in that more accurate entry and outcome criteria were specified. ECT was the most effective treatment at the end of four weeks. The percentage of patients with total remission or slight symptoms were ECT 71 per cent, imipramine 52 per cent, phenelzine 30 per cent, and placebo 39 per cent (Freeman, 1979). These differences were essentially maintained for ECT and imipramine at six months' follow-up. Rapid onset of action and greater efficacy in the first two months was reported for ECT.

In general, the available outcome literature indicates favourable outcomes in association with ECT relative to pharmacological methods, particularly in severely depressed patients (Turek and Hanlon, 1977; Crow and Johnstone, 1979; Freeman, 1979). Some concerns regarding possible problems with ECT will be discussed at the end of the chapter.

Before commenting further on the efficacy of physical treatments, it would be useful to consider the relationship between pharmacotherapy and psychotherapy. Both approaches to treatment are widely used in current clinical practice and frequently combined in a pragmatic fashion. However, little empirical evidence exists to guide the clinician in choosing between or in combining the two forms of therapy in any systematic fashion.

The next section of the chapter has two purposes: (1) to give a brief historical account of the thinking which preceded the systematic investigation of the effectiveness of drugs and psychotherapy in the treatment of depression, and (2) to describe the current evidence about the efficacy of this form of treatment in relieving the acute symptoms and problems associated with depressive disorders.

C. THE USE OF PHARMACOTHERAPY IN CONJUNCTION WITH PSYCHOTHERAPY IN THE TREATMENT OF DEPRESSION

1. Historical overview

The research on drugs and psychotherapy of the early and mid 1960's did not address the issue of the efficacy of psychotherapy compared to drugs, but mainly focused on how and when, if at all, drugs should be brought into the psychotherapeutic treatment of depressed patients (Weissman, 1978). A fairly extensive literature developed which contained astute clinical observations about the impact of introducing drugs into the psychotherapeutic relationship (Bellack and Rosenberg, 1966; Ostow, 1962; Sarwer-Foner, 1970). These clinical accounts paid little attention to diagnosis, type or quality of drugs or psychotherapy, length of treatment or the differentiation of possible outcomes under the different therapies. With few exceptions (Lorr et al., 1963) the literature seldom referred to controlled studies to test out the efficacy of combining drugs with psychotherapy.

At a time when the predominant opinion was negative for the combined use of drugs and psychotherapy, two psychoanalysts, Ostow (1952) and Sarwer-Foner (1960) argued for the addition of pharmacotherapy to psychological intervention. For example, Ostow recommended the selective use of drugs under certain conditions to help a patient who was too upset to participate in psychoanalysis or to protect a patient who might harm him or herself, or during periods when the attending physician was away. There were also several attempts at the time to examine systematically the hypothesised effects of drugs on psychodynamic process, such as the content of dreams, or the expression of anger (Bellack and Rosenberg, 1966; Klerman and Gershon, 1970; Sarwer-Foner, 1970).

Weissman (1978) notes that 'with increased experience and acceptance of pharmacotherapy for psychiatric disorders, concern about the effects of drugs on psychotherapy reversed to include questions about the effects of psychological factors on drug treatment. There were efforts to clarify the nature of these psychological effects. Questions were raised about the impact of such non-specific factors as the setting in which the drug treatment was administered, the physician's attitudes, or the patient's expectations (Fisher, 1970; Stone et al., 1975)' (P.1314).

This research trend, according to Weissman, had two main impacts: the placebo response in pharmacotherapy was found to be less than expected and there was clarification of the specific psychotherapies (e.g. Ellis, 1962; Beck, 1973) as distinct from the psychotherapeutic management of patients, or the psychological consequences of receiving treatment (for a detailed review of these see Weissman , 1978).

The late 1960's witnessed the gradual accumulation of evidence that drug therapies are effective in the treatment of acute depressive episodes and numerous attempts to delineate the nature of psychotherapies. It was inevitable that more sophisticated hypotheses about the interactions of drugs and psychotherapy would be developed. Thus, in addition to the deleterious effects of combining drugs with psychotherapy put forward originally, the effects were now hypothesised as feasible in either direction, pharmacotherapy on psychotherapy or vice versa. The interactions between treatments were regarded as being potentially positive as well as negative.

Klerman (1975) identified at least four types of potential effects of combined therapies. They are described below.

Negative effect of drugs on psychotherapy

Drugs obstruct psychotherapy by concentrating on symptoms rather

than aetiology, by amplifying reliance on the doctor and fostering dependency, by reducing the chances of gaining insight, hence recovery, by reinforcing the tendency for the doctor to use an easy way out and to be authoritarian, or by diminishing the patient's motivation to contribute actively to therapy.

Positive effect of drugs on psychotherapy

Drugs maximise the effects of psychotherapy by making the patient more accessible and by reducing anxiety, by increasing sleep and memory, or by restoring contact with the environment, thereby improving the capacity to form or maintain relationships. Drugs reduce distractibility and improve verbal skills.

Negative effects of psychotherapy on drugs

Increased anxiety produced by psychotherapeutic experiences counteracts the symptomatic recovery attributable to drugs.

Positive effect of psychotherapy on drugs

Psychotherapy solidifies the patient/doctor relationship and facilitates patient attendance and compliance with drug administration.

Klerman (1975) also pointed out that before prescribing drugs and psychotherapy, research should produce four types of evidence:

1. evidence for the efficacy of each treatment alone and then in comparison,
2. understanding of the mechanisms of action of the treatments,
3. verified concepts that bridge the two treatments and provide a foundation for the combined regimens,
4. evidence of the efficacy of the combination.

According to Weissman (1978), 'in the late 1960's much of this evidence was lacking. There were many controlled studies that demonstrated the efficacy of drugs in acute depressive disorders, partially verified hypotheses about their mode of action, and no research evidence for the efficacy of psychotherapy in depression (Klein and Davis, 1969). Combined treatments (drugs and psychotherapy) for the affective disorders were widely used and empirical research about their value was lacking' (P.1315). However, over the last ten years there have been exciting developments in the study of the psychology of depression and evidence as regards the efficacy of psychological interventions with and without drugs has begun to accumulate.

Having outlined the dominant state of thinking during the 1960's that precipitated the empirical investigation of drugs and psychotherapy, attention can now be shifted towards an evaluation of the available evidence concerning the efficacy of psychological approaches alone and in combination with drugs. An increasing number of experimental studies have appeared in the literature quite recently which indicate a role for psychological approaches without drugs in treating depressed patients, particularly behavioural and cognitive-behavioural techniques (Burgess, 1969; Rush et al., 1975; Rush et al., 1977). These and other psychological treatments demand a detailed review in their own right and will therefore be taken up in subsequent chapters. The next part of this chapter will focus specifically on the current state of evidence about the effectiveness of the combined use of drugs and psychotherapy in the treatment of depression. Only those studies incorporating some control comparison will be considered.

2. Evidence for the efficacy of drugs combined with psychotherapy in maintenance trials

In light of the empirical requirements for prescribing combined

therapies set out by Klerman, Weissman (1978) has noted that the most logical way to progress might have been first to test out the effectiveness of drugs and psychotherapy in the acute treatment of depressive disorders. However, the first controlled clinical drug studies including psychotherapy were started in maintenance drug trials by researchers who were interested primarily in the psychopharmacological treatment of affective disorders.

Although evidence had accumulated for the efficacy of antidepressants on symptom reduction in acute depression (see sections A and B of this chapter), there still remained the critical question of how to prevent relapse and/or improve social adjustment. By the late 1960's it was uncertain as to how long depressed patients should be maintained on drugs and if psychological techniques should be incorporated into drug therapy (Weissman, 1978; Klerman et al., 1974). Despite the lack of completed controlled trials of psychotherapy that included a homogenous sample of depressed patients (Lieberman, 1975), there was considerable clinical testimony at the time about the value of psychotherapy as the exclusive treatment of depression. Also, it had been a technique widely used in clinical practice, the rationale being that psychotherapy ameliorated problems related to impaired social and interpersonal circumstances. For these reasons, the National Institute of Mental Health supported three independent maintenance trials using drugs and psychotherapy.

In a study conducted in Baltimore, Covi et al. (1974) assigned neurotic depressive outpatients to receive imipramine, diazepam or placebo and to group psychotherapy conducted within a psychodynamic framework, but otherwise unspecified (90 minutes per week for 16 weeks), or to brief support (20 minutes each week). Taking final measures (i.e. 16 weeks or termination if earlier) on self-ratings of mood and symptoms, there was a main effect of

drugs, i.e. imipramine being superior, but not of psychotherapy.

The Philadelphia project (Friedman, 1975) assigned depressed outpatients to amitriptyline or to placebo and also to marital conjoint therapy (12 weekly one hour sessions) or to brief support (half-hour sessions every two weeks). This study was originally considered a maintenance trial since patients were followed through to recovery. However, in retrospect, it has obvious implications for acute treatment because patients were acutely ill during the initial weeks of the study. Taking only final assessment results which gain a one per cent significance level, there is evidence for a pharmacotherapy effect on both symptom ratings and on marital functioning, but only for the latter was there a desirable change related to the psychotherapy.

Klerman et al. (1974), Weissman et al. (1974), Paykel et al. (1975) conducted a study in two clinics (The Boston-New Haven Collaborative Project) of the specific and interactive efficacy of maintenance antidepressants and psychotherapeutic treatment on the depressive symptoms and social adjustment of outpatient female primary depressives. One hundred and fifty patients who responded favourably to a preliminary four to six week trial on antidepressant medication were assigned to a high or low psychotherapy contact condition. Responders then received an additional two months on drug maintenance.

After the initial one month of acute treatment and two months of maintenance drug treatment, patients within each of the two psychotherapy groups were randomly allocated to drug, placebo, or no tablet groups. The high contact group always included a minimum of one weekly individual supportive session which dealt with current adjustment issues. Drugs reduced depressive symptoms most effectively and as effectively with or

without psychotherapy, but drugs alone had little impact on social adjustment. In contrast, psychotherapy improved general social adjustment including work, communication, friction, anxious rumination, but had limited effect on the depression per se. Relapse rates were rather lower for drugs alone or in combination with psychotherapy (12 per cent) than with psychotherapy alone (16 per cent); 36 per cent relapsed in the low contact, no-tablet minimal treatment group. Of the initial cohort of 150 patients who completed at least two months of the maintenance phase, 11 dropped out and 33 relapsed before finishing the programme. Sociodemographic characteristics did not differentiate relapsers from completers.

Social adjustment measures were analysed for two periods: at the end of the four month drug maintenance phase and four months thereafter. Group adjusted differences were insigificant at four months, but considerably in favour of the high contact therapy groups (44 per cent improvement) over the low contact groups (28 per cent improvement) on completion of the eight month period. 'Submissive dependence' and 'family attachment' were the only factors yielding no differences. Drugs had no advantage over no drugs on social adjustment ratings nor did they interact with psychotherapy.

Becker (1977) underscores an observation noted by the investigators in that the potential effectiveness of the psychotherapy may be underestimated because of the initial two month drug phase. Many patients attributed their improvement mainly to medication and were less motivated to deal with social adjustment issues once they had obtained symptomatic relief. Becker also points out that 'most patients came from working class families and therefore may have lacked incentive for self-exploratory or actualizing procedures' (P. 134). Higher education and social class, a tendency to describe reality in psychological constructs, and high motivation for therapy

are among the sociodemographic factors typically associated with positive psychotherapy results. However, Klerman et al. suggest other possible biases that may have prejudiced ratings in favour of psychotherapy. Interviewers were not blind to the psychotherapy condition and no independent ratings by relatives or objective self-reports were gathered.

Given these biases, it is disconcerting to note that the size of the difference in social adjustment was not very large. After eight months' treatment the adjusted means differed by a mere 0.17 units on a five point scale. Moreover, inspection of the precipitating factors in those patients who relapsed (Paykel and Tanner, 1976) shows that neither the amitriptyline nor the psychotherapy protected against relapse associated with life events.

Nevertheless, in this investigation, as in the Philadelphia project, measurable changes were demonstrated on those functions which were the prime focus of the psychological techniques, though this seemed to have no beneficial effect on the symptoms of depression. Furthermore, the findings underscore the desirability of including social effectiveness criteria as well as symptom relief and relapse rates among outcome criteria.

The data from these studies (Covi et al., 1974; Friedman, 1975; New Haven/Boston Project) indicate a striking similarity in outcome. The three investigations demonstrated an effect for tricyclics in preventing relapse and diminishing symptoms and either little or no effect on social or interpersonal performance.

As regards the effect of maintenance drugs, all three studies show that in comparison to placebo or psychotherapy, tricyclics were more effective in preventing reoccurrence of symptoms and relapse. Symptom reduction appeared to facilitate some improvement in social performance but antidepressants generally did not help patients cope better with 'problems in living'.

The findings for the maintenance psychotherapies were also remarkably alike in that all of the investigations demonstrated an effect for psychotherapy in areas related to interpersonal relationships and 'problems in living', an effect that was negligible in relieving the acute symptoms of depression.

These investigations permit an evaluation of the results with respect to the various interactions of drugs and psychotherapy postulated by Klerman. This comment is best summarised by a quotation from Weissman's review:

> 'The New Haven-Boston study results, which found a psychotherapy effect only in patients who remained symptom-free, best supported the hypothesis that drugs have a positive effect on psychotherapy in that the symptom relief, produced more readily by drugs, rendered the patient more accessible to psychotherapy. There was no evidence in any of the studies for a negative interaction between drugs and psychotherapy. Therefore there was no evidence for the hypothesis that drugs have a negative effect on patients who had experienced symptom relief. Drugs did not make patients less interested in psychotherapy (this contrasts sharply with Becker's notion that social class may have contributed to an unwillingness to self explore after remission of symptoms) and did not lead to early termination or poor response to psychotherapy, nor was there any evidence for a negative effect of psychotherapy on drug response. Patients receiving psychotherapy were not symptomatically disrupted' (author's insertion, P.1318).

In summary, the studies of maintenance trials of combined treatments show that for depressed patients an argument can be made for combining drugs and psychotherapy techniques.

The results of these trials cannot be generalised to acutely depressed populations. Also, it may be said that the effects of both types of therapy appear to be independent, operating on different outcomes, and there are no reported negative interactions.

3. Evidence for the efficacy of drugs combined with psychotherapy in the acute treatment of depression

Favourable clinical accounts of the combined use of antidepressants and psychotherapy for acutely depressed patients have been reported in the literature (Lesse, 1960, 1966). These suggest that the availability of combined approaches has changed the management of depressive patients, resulting in less hospitalisation and less use of electroconvulsive treatment. However, there are three controlled trials to date which investigated the efficacy of drugs and psychotherapy in acutely depressed patients.

Daneman (1961) reported a double-blind comparison of imipramine plus psychotherapy versus a placebo plus psychotherapy in the treatment of private practice patients suffering from recurrent depressions (159 neurotic, 32 psychotic depressive reactions and four with depressions in organic brain syndromes). Psychotherapy consisted of 45 minute sessions of psychoanalytically oriented psychotherapy, once or twice a week over a period of between 30 and 90 days. Evaluations were made by the author (the only therapist) on a series of rating scales. Global judgements of improvement were the main source of data.

After one month, 79 percent of the imipramine psychotherapy subjects were said to be in 'full remission' compared to 7 per cent of the placebo-psychotherapy subjects. At two months, the comparable figures were 87 per cent for the drug and psychotherapy group compared to 10 per cent for the placebo plus psychotherapy group. The differences between the groups

were statistically significant, indicating the superiority of the combination of active drug plus psychoanalytic psycbotherapy over the combination of pill placbo plus psychoanalytic psychotherapy. On the surface it would appear that combining drugs and psychotherapy proved relatively effective.

However, there are several problems with this study. First, other forms of therapy were given to the patients in an uncontrolled manner (e.g. phenothiazines, family consultation). It is thus impossible to attribute the observed effects to the treatment under scrutiny. Second, at two months' evaluation attrition was as high as 49 per cent. Third, the use of one therapist limits the generality of the findings to other populations.

The use of couples therapy groups in conjunction with lithium has been compared with lithium plus supportive counselling in the long-term management of married bipolar patients (Davenport et al., 1977). This can be regarded as an investigation into the differential effects of treatment on acute symptoms as well, since the patients received weekly conjoint therapy sessions during an initial period of hospitalisation. Sixty-five patients were assigned arbitrarily to conjoint group therapy plus lithium or lithium plus individual supportive counselling. At approximately $2^1/_2$ years' follow-up, those in the combination group had more benign post hospital course than those given minimal support beyond lithium maintenance, i.e. the couples group had fewer rehospitalisations and marital failures than the lithium plus support group.

This study is important for two reasons. First, it represents the only comparative trial that involves diagnosed bipolar patients. Second the results tend to suggest that conjoint group therapy may have an impact on asymptomatic phenomena in manic-depressive patients.

In the most important study of its kind, Weissman et al. (1979) randomly assigned nonpsychotic, nonbipolar, acute primary major depressed outpatients to one of four treatment conditions: short-term interpersonal psychotherapy alone, pharmacotherapy, interpersonal psychotherapy plus pharmacotherapy and nonscheduled treatment. Interpersonal psychotherapy consisted of 'at least one weekly 50 minute session with the treating psychiatrist' (P.556), which focused on the social context of depression. Pharmacotherapy consisted of amitriptyline 100 to 200 mg dosage increased to 125 mg within a week, 'stabilised over the next 3 weeks and maintained for the next 12 weeks' (P.556). Combined therapy consisted of both treatments described previously. Nonscheduled treatment or 'psychotherapy on demand' consisted of allocating patients to a psychiatrist they could contact whenever they felt in need of care. No active treatment was scheduled, but the person could communicate by telephone, and 'if his or her needs were of sufficient intensity, a 50 minute session (a maximum of one a month) was scheduled' (P.556).

Of the 96 patients randomised into treatment, 81 were used in the analysis. Symptomatic failure was the main outcome criterion in the report, i.e. a Raskin depression score of 9 or more as rated by a clinical evaluator after eight weeks of therapy. The results show that the combination group had the lowest rate of failure whereas the highest failure rate was obtained in the nonscheduled treatment group. Patients in the active treatment groups demonstrated a significantly lower rate of failure than those in the 'psychotherapy on demand' group ($p = < .001$). The investigators note that while there were fewer relapses in the combined therapy group than in either psychotherapy or drugs alone, the difference was not highly significant ($p = < .10$). No significant differences in failure rates were found between the psychotherapy and pharmacotherapy

groups. Symptomatic failure occurred as early as the first week in the nonscheduled treatment group whereas there were no failures in the drugs and psychotherapy group before the twelfth week. No patients in the combined group had to be withdrawn at the prescribed eight week assessment period. Similarly, there were no symptomatic failures before week eight in either the drug or psychotherapy groups.

The investigators also point out that a small proportion of patients in the 'psychotherapy on demand' group were not symptomatic failures and reported satisfaction with this treatment. As the investigators note, 'it would be important to predict who are the substantial minority of patients who responded to this infrequent non-scheduled treatment' (P.558).

In order to clarify the amount of tolerance and efficacy of the various treatments, Herceg-Baron et al. (1979) in a follow-up report provide a detailed analysis of the attrition patterns in the treatment groups. 67 per cent of the patients in the combined treatment completed the full 16 weeks, compared with 48 per cent in psychotherapy alone, 33 per cent in pharmacotherapy alone, and 30 per cent in the non-scheduled group. Patients in the combined group were more likely to accept treatment initially than patients in the other treatment groups ($p = < .05$), more likely to complete treatment ($p = < .01$), and less likely to relapse. Patients treated with drugs and psychotherapy were less ill upon early termination than those receiving either drugs or psychotherapy alone or the non-scheduled treatment. This is a particularly interesting result inasmuch as those who dropped out of treatment for other than nonsymptomatic reasons did not receive further treatment. Furthermore, the investigators note that if patients dropped out of another treatment because of persistent symptomatology, they frequently asked for a combination treatment. For

example, 38 per cent of patients who initially refused psychotherapy subsequently accepted a combination of drugs and psychotherapy.

The research evidence for the efficacy of drugs and psychotherapy for acute treatment of depression in comparison to drugs or psychotherapy alone is meagre. Although Weissman et al. (1979), for example, conclude that the combination treatment was more effective than either treatment alone and delayed relapse, the difference in outcome did not achieve a satisfactory level of statistical significance. It should be noted, however, that any comprehensive evaluation of therapy and the relative value of different techniques should include criteria such as length of treatment and its acceptability to the patient, among others (Kazdin and Wilson, 1978). The findings on attrition patterns in the treatment groups in the Weissman et al. (1979) study suggest that combination treatment may be more acceptable to patients than either pharmacotherapy or psychotherapy alone. Moreover, once admitted to a course of treatment, patients may be more likely to complete a combined therapy regimen.

Thus, the combination of psychotherapy (group, individual and marital) in conjunction with drugs would appear to be an effective treatment for some depressive patients. Luborsky (1975) has suggested that the advantage for combined treatment represents a possible additive effect of the two treatments. This remains an empirical question. However, no matter what theoretical approach is used to explain the apparent efficacy of a combined approach, these studies suggest that psychological techniques are highly relevant to the pharmacotherapy of depression. Further research is necessary to establish firmly the efficacy of combined treatment and to answer questions about which type of psychotherapy to use with which drugs, for which depressive patients.

D. SPONTANEOUS REMISSION

Up to this point the chapter has focused on the efficacy of pharmacological treatments, ECT and pharmacotherapy in combination with psychotherapy in the management of depressive disorders. However, after nearly 25 years and much heated debate, it is disconcerting to find that there is substantial disagreement over the rate of improvement in neurotic illnesses in the absence of active treatment. This is a crucial issue because 'spontaneous remission' is thought to confound the success rates that are claimed for many forms of psychiatric treatment, especially psychotherapy (Eysenck, 1952; 1967; Bergin, 1971; Rachman, 1973; Bergin and Lambert, 1978). It is thus important to ask whether rates of success reported for active treatments profit by the effects of extraneous events or homeostatic mechanisms and, as Bergen and Lambert (1978) point out, whether there are reliable 'baseline' figures that represent improvement in neurotic patients who go without treatment.

While little is known about the actual process of 'spontaneous remission', several prominent researchers, notably Eysenck (1952, 1967) and Rachman (1973), have attempted to provide a baseline figure with which to compare the results of psychiatric intervention.

These authors obtained estimates based on a review of studies which examined the duration of neurotic illnesses not treated by psychotherapy. They argued that if a period of two years is accepted as a baseline measure (which is reasonable since psychotherapy does not usually last longer than two years and may involve less time), two thirds (66%) of neurotics show recovery or marked improvement without the benefit of psychotherapy, after a lapse of two years from the time that their disorder is identified, or they are hospitalised (Rachman, 1973).

Recognition that many neurotic disorders can be expected to remit spontaneously complicates the problem of determining to what extent recovery from an illness can be attributed to the specific effects of treatment. Moreover, there is some evidence that rates of spontaneous remission vary according to different diagnostic categories. Depression, in particular, is said to have one of the highest rates of spontaneous remission. For instance, Greer and Cowley (1966) carried out a four to six year follow-up study of 160 neurotic patients who were consecutive admissions to the Mawdsley Hospital. In an extensive investigation of different diagnostic groupings, they found that depressed patients had the shortest duration of symptoms (over 50 per cent were asymptomatic in less than six months) whereas patients diagnosed as obsessive reactions were found to have the longest duration of symptoms (five years or longer). Although Greer and Cowley emphasised premorbid personality as an important prognostic indicator, they concluded that the type of neurotic illness is also an important factor relating to outcome.

Bergin and Lambert (1978) tried, as Rachman (1973) did, to order rates of spontaneous remission according to type of neurotic illness and concluded that such an ordering cannot be accomplished by reference to the current research literature. According to these authors, 'it appears that anxiety and depressive neuroses have the highest spontaneous remission rates, followed by hysterical, phobic, obsessive compulsive and hypochondriacal disorders; but no study has attempted to describe recovery rates by diagnostic classification while holding constant other important variables such as degree of disturbance, type of onset, and past history of disturbance' (Bergin and Lambert, 1978, pp 147-148). They suggested that a complete revision of the classification of neurotic disorders is

required before any confidence can be placed in the ordering of remission rates.

Several authors (Bergin, 1971; Lambert, 1976; Bergin and Lambert, 1978) have contested the work of Eysenck and Rachman and established another baseline rate for spontaneous remission on the basis of reviews of studies of minimal treatment and no treatment outcomes. The median remission rate for untreated subjects, according to Bergin and Lambert (1978) is 43 per cent (range 18 - 67 per cent). In their view, 'a two thirds estimate is not only unrepresentative but is actually a most unrealistic figure for describing the spontaneous remission rate or even rates for minimal treatment outcomes' (Bergin and Lambert, 1978, p. 147).

The question of spontaneous improvement has stimulated considerable research and discussion. Whether the figure of 66% or 43% is accepted as the baseline estimate of spontaneous remission, it should be noted that there are average figures which, in fact, obscure considerable variation. In any event, the important issue at stake in this chapter is whether the improvement rates discussed in relation to the various treatments exceed those reported for spontaneous remission. It is clear that some studies on the efficacy of ECT (e.g. MRC Trial 71%) and to a somewhat lesser extent drug therapy (e.g. MRC Trial 52% for imipramine) fulfill this requirement.

It is worth noting, in summary, that some neurotic disorders are particularly responsive to unspecified extratherapeutic events, or non-specific treatment factors such as reassurance, attention and suggestion. They are thus sensitive to placebo and may show a high rate of spontaneous improvement. Patients in general practice who present with minor emotional disorders, frequently a mixture of mild anxiety and depressive symptoms, may be especially responsive to change. Wheatley

(1972), for example, has noted a placebo response rate of 50 per cent in various drug studies in general practice compared with active drug response rates of about 75 per cent.

Thus, it may be said that in order for specific treatments of depression to be regarded as effective, treatment response rates must be interpreted with caution, taking into account various estimates of spontaneous remission.

E. COMMENT

On the basis of this review, the following comments and conclusions can be made about the effectiveness of the physical methods of treatment of depression.

Clinical comparisons of tricyclics and placebo demonstrate the efficacy of pharmacotherapy. Inspection of the data from a recent survey of the literature and that compiled by Morris and Beck (1974) indicates that sixty-six out of ninety-nine (66 per cent) treatment comparisons in eighty-nine investigations show tricyclic antidepressants to be superior to placebo. Amitriptyline and imipramine were reported to be consistently more effective than placebo. A relatively small number of studies which compared the tricyclic derivatives, nortriptyline, protriptyline, doxepin and amoxapine with placebo reported significant effects in favour of the drugs.

When the effects of the conventional tricyclics (imipramine and amitriptyline) are compared with one another and/or tricyclic derivatives the majority of investigations report equivalent effects among the drugs in reducing depressive symptoms. Forty-eight out of seventy-six (63 per cent) treatment comparisons in seventy studies showed no significant

differences between tricyclic antidepressants. Similarly, direct comparisons of tricyclic derivatives with each other demonstrate an equivalent effect on symptoms. MAO inhibitors do not demonstrate the same degree of effectiveness as tricyclic medication. Morris et al. (ibid) report an overall success rate of 33 per cent for MAO inhibitors over placebo, though they point out that this figure is based on trials which included drugs that had not been approved by the Federal Drug Administration as of 1972. Even so, phenelzine has been found to be only as effective as placebo in men and less effective than placebo in women (MRC Trial, 1965). While early studies have been criticised for inadequate dosage and duration of MAO inhibitor treatment (Tyrer, 1976) and some researchers have suggested that it may be particularly effective in 'atypical' depressions (Sargent, 1961), the findings clearly indicate that tricyclic drugs are to be preferred in the treatment of severe depressions.

The introduction of the tetracyclic substances, namely Maprotiline (Ludiomil) and Mianserin (Bolvidon), in the treatment of depressive illness is a potentially valuable addition to the everwidening range of pharmacological therapies. These may be particularly useful in patients with a low tolerance for unwanted anticholinergic effects. Several studies report that tetracyclics also appear to show a more rapid onset of action than tricyclics. Nevertheless, the bulk of evidence indicates that tetracyclic drugs offer little therapeutic advantage relative to tricyclic medication. Further research is necessary to confirm the findings that they are effective antidepressants.

Lithium carbonate is effective primarily in the treatment of bipolar illness. The value of the drug as an acute anti-manic agent and as prophylaxis in the longer term treatment of manic-depressive illness is

well-accepted. The more controversial role of lithium lies in its use as an acute treatment of depressive episodes, although recent controlled studies in the United States support the use of lithium as an acute antidepressant (Goodwin et al., 1972; Nayes et al., 1974; Baron et al., 1975; Mendels, 1976). European authorities have considered the drug of little value in the management of acute depressive episodes (Schou, 1971, in Worral et al., 1979). Further research must be conducted in order to establish firmly its efficacy as an antidepressant.

There is evidence that electroconvulsive therapy (ECT) is an effective treatment in severe depressive disorders. Reasonably well-designed and controlled investigations suggest that ECT may be more than the most effective pharmacological methods and reduces symptoms more rapidly. There is a current body of opinion that in general ECT may cause brain damage (Friedberg, 1977) and in particular lasting memory deficits (Squire and Chase, 1975). Also, it has been suggested that the procedure causes cognitive deficits which have escaped detection with the currently available psychological tests (Crow and Johnstone, 1979). Nevertheless, the data show that ECT must be regarded as one of the more effective front-line physical methods of treatment for severely depressed inpatients.

Though it is undisputed that available somatic methods of treatment work and for the short-term treatment of depression may appear to be cost efficient, there are some disquieting facts and deficiencies in the therapeutic response of depressed patients. Most investigations report that physical methods of treatment have an average failure rate of approximately 20 per cent (Blackburn, 1977). For example, in the MRC Trial (op. cit.) where ECT, imipramine, phenelzine and placebo were compared, if the mean response to treatment is calculated, it is clear

that 80 per cent of the patients had been discharged by the end of the 24th week. Interestingly, 45 per cent of patients on placebo improved after four weeks. Different investigations of the effects of ECT report similar percentages of failures: Angst (1961) 20 per cent; Carney et al. (1965) 17 per cent; Stromgren (1973) 24 per cent.

Inspection of the literature on tricyclics reveals a somewhat higher failure rate. Beck (1973), for example, estimates that between 35 and 40 per cent of patients who receive a first course of tricyclics do not improve during treatment. The data is consistent with the common clinical observation that a fair proportion of patients so treated fail to demonstrate a satisfactory or sustained clinical response.

This problem is highlighted by Quitkin et al. (1976) in their review of five follow-up studies which show that, in follow-ups of six months to two years after the acute symptoms of depression had been treated by ECT or tricyclics, relapse rates varied from 12 to 32 per cent on drugs and 28 to 85 per cent on placebo or no pill. On the face of it, it appears that for those patients who do improve, recovery may be slow or incomplete and there are frequent relapses (Blackburn, op cit). Furthermore, several studies have shown that drug treatment produces symptom relief but fails to improve social adjustment (Park and Imboden, 1970; Tanner et al., 1975).

The type of review presented in this chapter permits the formulation of conclusions with respect to the efficacy of the physical methods of treatment of depression and is helpful in describing research trends. However, advances in experimental designs, epidemiological techniques and the specification of reliable diagnostic criteria may encourage future researchers to concentrate on the specific interrelationships that are obscured by an overall summary of research trends.

Various selection factors, i.e. type of depressive disorders,

availability and type of psychiatric services may greatly interact with treatment effects. For example, a sizeable proportion of the findings reported here are based on samples of depressed inpatients. As inpatients represent a relatively small and highly selective sample of depressively ill people who tend to present with serious, psychotic, suicidal, therapy resistent and chronic depressions (Helmchen, 1979), it is doubtful that treatment response rates obtained on such a restricted sample can be applied with confidence to depressed outpatients. At the very least, the generalisation possibilities of research results gained from depressively ill people in the hospital are limited to an unknown degree.

It is therefore necessary to conduct more investigations of the effects of therapy on outpatients because (1) by far most depressed patients are treated in outpatient settings (Akiskal et al., 1978); (2) those patients who are hardly ever seen in a hospital are also treated as outpatients with depressions, and (3) outpatient treatment is carried out under very different conditions from hospital treatment (Porter, 1970).

The acute-chronic illness dimension may be an important factor related to observed treatment effects. Klerman and Cole (1965) reported that 46 per cent of acutely ill inpatients supposedly improved on placebo. Similar improvement was found in only 16 per cent of chronic inpatient and outpatient samples. In their statistical review of controlled trials of imipramine, Rogers and Clay (1975) compiled data which show that 34 per cent of acutely ill patients improved on placebo while only 20 per cent of chronic patients responded to the inert substance. Moreover, only one out of six investigations involving chronic populations reported significant results in favour of the drug.

There appears to be some evidence that other patient characteristics (e.g. age, sex) may significantly interact with effects of treatment. For

example, severely depressed older women were significantly more effectively treated with amitriptyline than with imipramine, particularly with regard to symptoms of depressed mood, sleep disturbance, and agitation (Hordern et al., 1963; Hordern et al., 1965). The authors suggest a strong specific interaction between the patient's age, sex and administered treatment.

Brown and Harris (1978) hypothesise that socio-environmental factors sensitise an individual to developing depressive symptoms and it is possible that these too contribute greatly to observed treatment effects, particularly for those individuals treated as outpatients. Recently, it has been argued that the well-documented sex difference in depression may be due to a sex difference in susceptibility, in precipitating factors, or in both. For instance, Radloff and Rae (1979) found that women were exposed more often to more of the factors that relate to depression, though the factors related to depression similarly for men and women. Since the sex difference was still evident after patients were matched on the factors, it is possible that there is a sex difference in susceptibility. Thus both learned factors and biological sex differences might contribute to susceptibility to depression, hence to treatment effects. A related area is the literature on the role life events, especially those viewed as stressful, may play in the genesis and maintenance of depressive disorders (Paykel, 1974).

Several investigators have called attention to potentially significant differences in the chemical and action characteristics of drugs, e.g. time of onset of effect (Snow and Rickels, 1964; Sandfer et al., 1965) and individual biochemical and physiological factors, e.g. individual differences in drug uptake by the brain (Haydu et al., 1962). However, it is only recently that researchers have attempted to couple these areas systematically with investigations of clinical drug efficacy by measuring 'blood levels'

of a drug as opposed to dosage. Systematic attention to these data as well as those previously mentioned may dispel much of the contradiction present in the drug literature.

The results of the drug and psychotherapy studies, in general, support the efficacy of tricyclic medication in reducing depressive symptoms. Conventional psychotherapies (Daneman, 1961; Covi et al., 1974; Friedman, 1975; Klerman et al., 1974; Weissman et al., 1974) appeared on the whole to have no discernible impact on depressive symptoms, though marital therapy (Friedman, 1975) and individual psychotherapy (Weissman et al., 1974) seemed to improve aspects of social adjustment.

The combination of tricyclic drugs and interpersonal psychotherapy in the treatment of acutely depressed outpatients appears to have had a greater impact in reducing depressive symptoms compared to tricyclics alone and interpersonal psychotherapy alone (Weissman et al., 1979).

These data suggest that combining drugs with psychotherapy may widen the spectrum of phenomena affected and that there may be an additive effect when these two forms of therapy are combined systematically. To date, there is no evidence for any negative drug and psychotherapy interactions (for an excellent review of combinative approaches see Hollon and Beck, 1978).

To conclude, there is a need to provide individualised treatment regimens for depressed patients. A chemical agent that appears to produce an antidepressant effect in one person may not produce the same effect in another depressed individual. A given patient may respond differently to the same drug at different times. This is likely to be the case for psychotherapies as well when used alone or in conjunction with drugs.

More research is needed not only with the physical treatments mentioned, but also with the effects of specific psychotherapies combined with physical treatments in relation to specific types of depression.

F. SUMMARY

This chapter has reviewed studies concerned with the efficacy of pharmacotherapy, electroconvulsive therapy, and the combined use of pharmacotherapy and psychotherapy in the treatment of depression. While there is strong evidence that physical treatments work, biochemical theories derived from the study of the pharmacological action of drugs have not produced the long-awaited discovery of a distinct relationship between depression and a specific biochemical event (Hollon and Beck, 1979; Blackburn, 1977). This has contributed to an increased interest in the psychological causation of depression (for reviews see Akiskal and McKinney, 1975; Blaney, 1977) and undermined the degree of confidence that can be placed in a purely biological model of depressive disorders.

The next chapter examines the most prominent psychological theories and techniques which are shaping our understanding of the development and maintenance of clinical depression.

CHAPTER THREE

PSYCHOSOCIAL APPROACHES TO DEPRESSION

A. INTRODUCTION

Utilisation of any set of techniques such as drug therapy, psychotherapy, a combination of these, or behaviour therapy to reduce emotional disturbance or alter personality assumes some model of the nature of man. The model includes assumptions about motivational factors, the processes that contribute to and influence attitudes and behaviour, the extent to which events shape a person's life, and the degree to which the influence of such events can be changed (Craighead et al., 1976). A theoretical framework also determines the manner in which behaviour is conceptualised, the causes to which behaviour is attributed, and the systems of intervention deemed appropriate to alter behaviour and relieve concomitant emotional strain.

Numerous aetiological models of depression have been put forward. Chapter two reviewed outcome studies on the use of drug therapy in the treatment of depression. The demonstrated efficacy of antidepressant drugs together with increased understanding about their mode of action has stimulated much research into the development of biochemical theories of depression. These include the association of clinical depression with a functional deficiency of biogenic amines at central postsynaptic receptor sites (Schildkraut, 1965; Coppen, 1967; Tissot, 1975), cell electrolytes imbalance (Shaw, 1979; Van Praag, 1978) and abnormalities of neuroendocrine function (Van Praag, 1978). Others have proposed that depression

may result from genetic deficits (Leonhard et al., 1962; Angst, 1966; Perris, 1966, 1968), internal psychological processes (Abraham, 1911; Freud, 1914; Rado, 1928; Fenichel, 1945; Jacobsen, 1946; Bibring, 1953), maladaptive interactions between the organism and the environment (Ferster, 1965, 1966, 1973; Lazarus, 1968, 1979; Seligman, 1976; Lewinsohn, 1976; Beck, 1973, 1976) and social deprivation (Brown and Harris, 1978).

Despite this diversity in causal theories, the phenomenology of depression has been described in remarkably consistent terms since the time of Hippocrates (Zilboorg and Henry, 1949). As cited earlier, depression has been traditionally regarded as a mood disorder but the clinical picture also includes marked changes in cognitive, behavioural, motivational and vegetative functions (Burns and Beck, 1978). In a bold attempt to collate the data from these diverse sources, hence to establish conceptual inroads among the different aetiological theories, Akiskal and McKinney (1975) proposed a multi-component model of depression. They maintain that any number of different events allied with areas of specific vulnerability - psychological, social, physiological and biochemical - can combine to produce a common psychobiological process that may be identified as a depressive illness. According to these researchers, no matter what psychosocial stresses contribute to the onset of depression, once the biological integrity of the organism is disrupted, the process becomes autonomous and virtually impregnable to psychological interventions. At this stage, the most immediate problem confronting the clinician is to decide which of the somatic therapies to use in order to stop the illness process.

In view of the efficacy of antidepressant medication, and until

recently, the conspicuous lack of demonstrated benefit from psychosocial interventions on depressive symptoms (Covi et al., 1974; Friedman, 1975), it is hardly surprising that the management of clinical depression had remained the province of the physical treatments. Indeed, when Akiskal and McKinney proposed their model, the available literature amply supported their pessimistic evaluation of the effectiveness of psychological approaches. Enthusiasm generated by case studies using behavioural methods (e.g. Burgess, 1969; Rush et al., 1975) had been justifiably dampened by a persistent lack of controlled investigations. Hollon and Beck (1979) note 'that as late as 1970 there existed no published study in which any type of psychotherapy had been shown to be more effective than either alternative treatments or no treatment conditions in any controlled comparisons with a homogeneously defined sample of depressives'. (P. 154)

This unsatisfactory state of affairs has changed considerably with the burgeoning interest in the psychological causation of depression (Blaney, 1977). The past decade has witnessed a healthy growth in empirical studies that are beginning to provide support for several cognitive and behavioural models of depression (Beck, 1967, 1973, 1976; Lewinsohn, 1974; Seligman, 1976; Rehm, 1977). In addition, findings from several comparative outcome studies indicate that some psychological therapies have a place in the treatment of some outpatients who present with mild to moderately severe depression. Particularly intriguing is the potential role for behavioural or cognitive-behavioural interventions and the findings of one of the studies (Rush et al., 1977) suggest that such an approach might be more effective than treatment with one of the most potent tricyclic antidepressants (imipramine hydrochloride).

Chapter two also covered the literature on the use of tricyclic antidepressants in conjunction with traditional forms of psychotherapy in treating depressed patients. It was argued that the combination of pharmacological and psychological treatments offers a potentially unique advantage in the treatment of some depressions. Although it is too early to specify for which types of depression this approach might be indicated, controlled outcome studies suggest that the combination treatment is generally more effective than the exclusive use of either treatment alone, especially when multiple outcome criteria are considered (Klerman et al., op cit.; Weissman et al., op. cit; Herceg-Baron et al., 1979). This advances the likelihood that biologically and psychosocially oriented clinicians will work more closely in planning treatment programmes for depressed patients.

But to what extent is depression a psychosocial phenomenon? In what ways have psychosocial models of depression been translated into specific, remedial interventions? How do such approaches compare with existing methods of treatment?

It is beyond the scope of this chapter to review the complete range of psychosocial theories and therapies of depression. For this thesis it is important to discuss the models which have had a major impact on the conceptualisation of depression and the therapeutic procedures derived from each model.

As mentioned previously, it is recognised that biochemical models may play a central role in explaining the wide variation of depressive symptomatology. However, an enormous literature, stimulated by studies of the mode of action of the various somatic treatments, prohibits inclusion of biological models in this review (see Paykel and Coppen, 1979).

The chapter is therefore broken down into three main sections: psychoanalytic, behavioural and sociological theories. The review describes these major theories of depression and considers evidence in support of each model, comparing the approaches with one another.

As the central argument of the thesis rests upon experimental support for a cognitive model of depression, a detailed description of Beck's theory and evidence bearing on the relationship between cognitive factors and depression is reserved for the final chapter of the literature review.

B. PSYCHOANALYTIC THEORIES

1. Overview

The early analytic writers viewed depression as a psychological disorder caused by the inadequate resolution of intrapsychic conflicts experienced during the psychosexual stages of childhood. Initially, they stressed the concepts of orality, narcissism, and ambivalence, but later, the focus shifted and increasing emphasis was placed on the roles of the ego and social relations.

Systematic understanding of the dynamics of depression began with Abraham's observation that the loss of a loved object is common to both mourning and melancholia, or clinical depression (Abraham, 1911, 1916). He thought depression was a consequence of regression to the oral level of libidinal development, with the typical oral characteristics of intolerance and envy, increased egocentricity and ambivalence. Aggression prevents the depressed patient from receiving or expressing love which leads to feelings of deprivation. Abraham compares clinical depression to a form of dying. He also discusses depression in terms of an obsessional neurosis (Abraham, 1924).

The depressed patient, like the obsessional neurotic, expends much libidinal energy in an effort to repress latent hostility. When confronted with the loss of an object, he projects the hostility outwards and concludes that he is hated by those around him. Outward-directed hostility is further generated by his acute sense of impoverishment which, in turn, produces guilt, anxiety, and self-reproach.

Freud (1917), in his celebrated paper 'Mourning and Melancholia', likened clinical depression to normal grief. Both may happen as a reaction to the loss of a loved object but in depression the response to the loss, seen as separation, frustration, and disappointment, remains in the realms of the unconscious. Thus, in normal grief, facing reality leads to an adaptive, redistribution of libidinal energy towards other objects. On the other hand, in clinical depressions the conflict of ambivalence is internalised. As Blackburn (1972) notes 'the intense self accusations are expressions of hate towards the ambivalent internalised object (as opposed to Abraham's view of hostility directed against the self). Freud explained this as the narcissistic identification of the ego with the object through introjection, a regression to the oral stage of libidinal development' (author's insert, P.56).

Rado (1928) also described depression in intrapsychic terms but advanced the theory of identification a step beyond the Abraham-Freud formulation. The original model had as its cornerstone the incorporation of the lost or frustrating object in both the ego and superego (ego-ideal). Rado suggested that depression may be caused by the rebellion of the ego against a cruel superego. Rather than an attack from the superego, depression comes from the ego's struggle

to regain the superego's approval through self-castigation, just as the child in his early efforts tried to win the love of his parents by self-punishment. He explained that the child had split his parents into 'good' and 'bad', assigning the bad, aggressive parts to his age and the good, loving parts to his superego. Depression is an attempt to destroy these bad parts of the ego, which, when purged, can unite with the superego in reciprocal love (Ashworth, 1976).

In Rado's view, depression is an intense yearning for love. Love is essential for the maintenance of self-esteem. Since the ego of the depressed person is grossly deficient in critical evaluation of his own achievement, he relies upon the approval of others to satisfy his overwhelming need for love. This description represents a significant departure from early psychoanalytic writings. According to Ashworth (ibid), 'It indicated a shift in emphasis from id to ego. In a later paper (Rado, 1951), he abandoned the concepts of introjection and narcissism and regarded the self torment of melancholia as purely an ego phenomenon wherein the ego is trying to regain the lost loved object, and therefore his self-esteem, by submitting his rage as abandonment to . . . fear and thus retro-flexing the rage inward.' (P.8)

Fenichel (1945) agreed with Abraham and Freud about the importance of hostility in depression but was quite explicit about what he termed 'the struggle for the maintenance of self-esteem' (P.139). He stated that 'normal' sadness as well as the melancholic forms of depression have in common a decrease in self-esteem. The clinical differences are viewed as stages in the course of the struggle to regain the lost self-esteem by various adaptive mechanisms. Fenichel argued that

depressed people respond to frustration with anger which they deny. The self-reproach of the depressive patient consists of the discharge on the ego of the aggression unconsciously felt for the lost object.

Bibring (1953) was the first to conceptualise depression strictly in terms of ego psychology, focussing on object loss and its consequences on the ego. His neoanalytic theory, a major conceptual break with the Abraham-Freud model, views depression as an ego state, an affect completely divorced from the oscillations of the aggressive drive. Bibring postulated that depression need not result from conflict or an attempt at synthesis between the ego and superego. Rather, depression may occur when the ego is cognizant of its goal and simultaneously aware of its helplessness to attain it. Self-esteem collapses because the ego feels unable to live up to its aspirations and depression is an emotional expression of the ego's impotency. Depression, in other words, develops when an individual cannot match his ego ideals - the desire to be worthy and loved, the striving to be secure, powerful and competitive, the aim to be good, kind, and compassionate. Although hostility may be generated by object loss, or obstacles that block the path to cherished goals, it is a secondary phenomenon in Bibring's model. Early childhood traumatic experiences as well as frustration of attainment of aspirations predispose individuals to depression.

Jacobsen (1953) takes up Bibring's point about loss of self-esteem in depression. She introduced the concept of self and object representations which develop during infancy into realistic and stable images in the ego, and also serve as the foundation for the ego ideals.

This brief overview of psychoanalytic theory may give an idea of the main contributions to the field of depressive illness (for detailed reviews see Greenacre, 1953; Becker, 1974). The phenomenon of low self-esteem in depression was a key factor in all these models: it was seen as a consequence of anger turned against the self, or a manoeuvre to appease a cruel superego, or the awareness that cherished goals are unobtainable. The emphasis shifted away from internal psychological processes - intrapsychic divisions, narcissism, orality, ambivalence - towards external events such as early familial experiences and the need for social approval. The neo-analytic school, in other words, rejected the view that depression results from an aggressive drive generated within the unconscious. Instead, it is seen in terms of a conscious cognitive factor (loss of self-esteem).

Although many of the early psychoanalytic conjectures are untested and untestable, they have been of considerable heuristic value and suggest hypothetical constructs for scientific study. Loss-separation and hostility are two such constructs that have been investigated.

2. Loss and separation

Since the concept of loss and separation serves as the corner-stone of psychoanalytic thought, it is here discussed separately.

In his well-known paper 'Anaclitic Depression', Spitz (1946) described a syndrome occurring in hospitalised infants who had been denied maternal contact. He observed that many of these infants failed to thrive, that their mortality was high , and that they were withdrawn and unresponsive and appeared to be depressed. Aetiologically, race, sex, age, developmental and intellectual level seemed irrelevant,

the one significant precipitating factor being that the infants were forcibly separated from their mothers during the second six months of life.

Bowlby (1960) confirmed Spitz's findings in his own studies on the mother-child relationship. He postulated that anaclitic depression was a consequence of the separation of the infant from its mother. Moreover, he argued that the depression was a real state of grief in the child. Bowlby went on to describe an initial stage of 'protest' comprised of agitated behaviour, soon followed by withdrawal and retardation (depression). In addition to emphasising the parallels between anaclitic depression in the child and normal grief in the adult, Bowlby also advanced the hypothesis that the loss of a loved object in childhood makes an individual vulnerable to pathological grief reactions in later life.

Although there is considerable disagreement about whether the syndrome described by Spitz and Bowlby is analogous to the clinical state of depression in the adult (O'Connor, 1956; Whoolton, 1959) their findings have stimulated research into the effects of early environment in depression. This is exemplified by animal studies of the loss-separation model (Harlow, 1955, 1971; Senay, 1966; Karfman, 1967; McKinney, 1969, 1971; Kaufman et al., 1967) and studies which show a positive relationship between childhood bereavement and depression (Brown, 1961; Munro, 1965; Beck et al., 1963; Denneby, 1966; Forrest et al., 1965).

Other evidence that early loss can make people vulnerable to later loss is reported by Levi et al. (1966) in which a particular cluster of both early and recent loss was noted in suicide attempters, relative to a control group of non-suicidal psychiatric patients.

However, Sethi (1964) failed to replicate this result in depressed patients. Buchnell (1970) compared depressed and non-depressed inpatients and found that severely depressed patients reported significantly more early death of mother than moderately severe depressives, though the incidence of parental death before age 20 did not differ between the two groups.

Granville-Grossman (1968) in their review of the relevant literature on depression, point out that the term 'parental deprivation' has been used indiscriminately to mean childhood bereavement, as well as separation from parents for other reasons, and maladaptive relationships with parents. Moreover, the term usually refers to maternal deprivation, even though some writers (e.g. Batchelor and Napier, 1953) have discussed the significance of the loss of a father figure. This confusion of terms may account for some inconsistencies in the loss-separation literature. Many investigations have failed to show a clear relationship between childhood bereavement and depression (Perris, 1966; Oltman et al., 1951; Gregory, 1939, 1962; Pitts, et al., 1965). If early loss is related to depression, there should be a higher incidence of early loss in depressed patients relative to the general population or other psychiatric groups, but again Granville-Grossman (1968c) concluded that the evidence for this hypothesis is far from compelling.

In summary, the loss-separation model derived from psychoanalytic theories offers a means of testing an aetiological theory (object loss) and also has the potential for delineating what other variables in the social field influence the development and maintenance of depression. While empirical support for the association between early bereavement and depression has been equivocal, the model has had a profound impact

on theory and practice. It has contributed significantly to the development of behavioural theories and the study of life events and social factors in depression. These areas will be taken up in more detail later in the chapter.

3. Hostility

The classical psychoanalytic writers (Abraham, 1911; Freud, 1917) viewed depression as a consequence of the inward turning of the aggressive instinct that, for various reasons, is not directed towards the appropriate object. The individual experiences retroflected anger in response to the real or symbolic loss of an ambivalently loved object. The theory is described in metapsychological terms and does not lend itself readily to empirical investigation. Nevertheless, as the mainstream of early analytic thought on depression views retroflected anger as crucial, attention is here directed to selected studies on hostility.

The anger-turned-inward model of depression has not received much empirical support. For example, if depression is supposed to be a product of converted internalised anger, then levels of overt anger and overt depression might be expected to be inversely correlated, whereas increases in the overt expression of anger should covary with clinical improvement. In one clinical study (Friedman, 1970), measures of overt hostility and 'covert' hostility (e.g. guilt) were positively correlated with one another and negatively correlated with improvement. Thus, the exact opposite of the predicted relationship held true. Klerman et al. (1970) also found that reduction of hostility towards external objects did not correlate with clinical improvement.

While many studies have failed to demonstrate a causal relationship between the direction-of-hostility theory and depression (Akiskal and McKinney, 1975; Hollon and Beck, 1979), there is strong support for the role of aggression in the phenomenology of depressive illness. For instance, in order to measure extrapunitiveness (anger against others) and intrapunitiveness (anger against self) Foulds (Foulds et al., 1960; Foulds, 1965) devised the 'Hostility and Direction of Hostility Questionnaire (HDHQ)'. Using this instrument, investigators (Mayo, 1967; Philips, 1971; Blackburn, 1975) have consistently shown that general hostility (extrapunitiveness and intrapunitiveness) is higher than average during depression and diminishes to a normal level after recovery, the greater change being in intrapunitiveness with only a slight change in extrapunitiveness.

Clinical investigations have frequently underscored the importance of punitiveness, especially intrapunitiveness, as a key factor in syndrome depression. Grinker et al. (1961) conducted a factor analysis of the phenomenology of depression and noted that five components described the feelings and worries of depressed patients. One of these (factor III) appeared to describe attributes of guilt over wrong-doing by the patient. Feighner et al. (1972) and Spitzer (1978) include 'feelings of self-reproach' among their research diagnostic criteria for depression. Recently, Blackburn (1979) used several depression scales and a self-rated hostility measure (HDHQ) to investigate the relationship between hostility and depressed mood. Nineteen patients were investigated in a follow-up design. She found significant intercorrelations among the mood measures which decreased over time, as did two of the four hostility

measures ('intrapunitiveness and direction-of-hostility').

The traditional notion that depressed patients show less overt anger than nondepressed people has also been challenged. Although depressed patients may display little anger in the presence of high status figures such as professional helpers (Weissman and Paykel, 1974), hostility is quite often intense and overtly expressed towards significant others (Weissman et al., 1971). That depressives may selectively control overt expressions of hostility is consistent with the concept of ambivalence which, according to the classical analytic school, plays a central role in the aetiology of depression. On the other hand, several researchers have argued that there may be a subgroup of depressed patients, i.e. 'hostile depressives', characterised by a simultaneous presence of depressive symptoms and overtly expressed anger (Overall et al., 1968; Lazarus, 1968; Paykel, 1971).

Masochistic behaviour, commonly regarded as a consequence of retroflected hostility, has been explained within the context of learning theory. For example, Forrest and Hokanson (1975) demonstrated a reduction in physiological arousal following preference for self-injurious behaviour ('masochism') in a dyadic interactional experiment. Depressed and nondepressed college students were given a choice of responding to another subject's response by (1) delivering a shock to the subject, (2) administering a less severe shock to themselves, or (3) giving a friendly signal to the subject. The depressed patients preferred the self-shock system and evidenced reduced physiological arousal, labelled by the experimenters as an indice of perceived relative personal safety. However, Forrest and Hokanson were able to modify the preference for self-injurious

behaviour and physiological arousal reduction in both the depressed and non-depressed groups. Response contingencies were subsequently arranged so that the initially non-selected responses, i.e. expressed hostility using countershock for depressed subjects and self-shock ('Masochism') for the non-depressed subjects, became differentially rewarded, thus eliciting a friendly signal from the other subject. The new contingencies were in operation over ninety trials. Depressed and nondepressed subjects shifted behavioural choice and physiological arousal reduction to the differentially rewarded response.

The investigators postulated that masochistic behaviour, rather than being a function of the anger-turned-inward model, may be maintained by differential reinforcement, with low levels of self-injurious behaviour serving to pre-empt harsher, punitive responses from others. The selection of an alternate response (self-injurious behaviour) in favour of an aggressive strategy (shock delivery) was interpreted within Bandura's social-learning model. Other studies (Hokenson et al., 1963, 1968) have shown that women tend to adopt similar strategies when interacting with men, as do students interacting with lecturers.

According to these findings and those of Weissman and Paykel (op. cit), the expression of hostility may be regulated by perceived environmental contingencies and the social context in which the individual behaves.

If depression results from converted internalised anger, then predominantly hostile themes should be evident in the dreams and fantasies of depressed individuals. However, investigations of manifest dream content have shown an excess of themes of loss

and failure instead of aggression in both ill and remitted patients (Beck and Horvich, 1959; Beck and Ward, 1961; Hauri, 1976). Beck (1963) also examined the content of free association fantasies of depressed patients during psychoanalysis and found a tendency for patients to report themes of failure and low self-esteem rather than hostility. He concluded that the findings could be interpreted as demonstrating a belief in personal incompetence rather than retroflected hostility. As will become clear, this position is more in line with neoanalytic concepts and a cognitive theory of depression.

The Abraham-Freud formulation also predicts that some depressed individuals may 'punish' themselves indirectly by rejecting opportunities to experience pleasure, withdrawing from family and friends, and by 'resisting' progress in therapy. This apparent motivational process has been referred to by clinicians as an unconscious wish to fail. While it is true that depressives demonstrate performance deficits on certain tasks (Miller, 1975), such deficits need not be attributed exclusively to motivational of physiological inhibitions. The findings of success-failure experiments suggest that reduced performance may be a consequence of lack of confidence, indecisiveness, and negative predictions about performance (Beck et al., 1962). Two investigations (Klein and Seligman, 1976; Loeb et al., 1971) have demonstrated that following feedback about success, depressed subjects experienced an increase in mood and performed better on subsequent tasks. Again, these data raise further challenges to the internalised anger model and could be more parsimoniously interpreted as consistent with a cognitive theory of depression. This model predicts that

depression is caused by 'negative expectations' rather than an unconscious desire to fail.

The retroflected anger theory, in summary, offers a creative and intuitively appealing explanation of the development of clinical depression. It has generated hypothetical constructs for elucidating aetiological factors and has been helpful in delineating the phenomenology of depressive illness, in particular the role of hostile affect and behaviours. However, there is not much empirical support for the theory. Findings on the association between overt hostility and depression and the inverse relationships between overt hostility and improvement, the regulation of self-injurious and aggressive behaviours in response to recognised social status differences and outcome expectancies, the excess of themes of loss and failure in dreams and fantasies, and the positive response to feedback about effective performance are inconsistent with the anger-turned-inward hypothesis. Instead, the data appear to be more readily accommodated within a cognitive-motivational model, whereby depressed people believe themselves to be failures and doomed to lifelong deprivation.

4. Controlled treatment studies

Psychoanalytic thought is undoubtedly one of the leading influences in the psychological treatment of depression. Explorative psychotherapy, grounded in both clinical and neoanalytic writings has been described extensively in the clinical literature (e.g. Wolberg, 1954). Orthodox psychoanalytic therapy is a prolonged and intense process. The principle components of therapy include free association, catharsis, and interpretation. Change and reconstruction of personality is supposed to come through the release of repression, the discharge of painful effects (e.g. anxiety and hostility) and

the resolution of transference neurosis. Neoanalytically oriented psychotherapists usually attempt a briefer treatment through a flexible application of a variety of techniques (for excellent reviews see Wolberg, 1954; Bergin and Strupp, 1972).

The ultimate goal of most forms of traditional psychotherapy is to provide a therapeutic situation in which the patient can achieve insight into the neurotic nature of his relationships and symptoms, the initially unconscious fantasies that lie behind these, and their origin in his childhood experiences (Nemiah, 1961). In recent years psychotherapists have focussed more on the 'here and now' and the amelioration of impaired social and interpersonal circumstances (Whitehead, 1979).

The first controlled studies of the effectiveness of traditional psychotherapeutic approaches were conducted by psychopharmacologists interested in the combined use of tricyclic antidepressants and psychotherapy. The previous chapter reviewed this literature. Three separate studies have demonstrated that traditional psychotherapeutic interventions are less effective than drug therapy alone or that combined psychological/drug therapy interventions are no more effective than drug treatment alone in terms of reducing syndrome depression or preventing relapse (Covi et al., 1974; Friedman, 1975; Klerman et al., 1974). There is one investigation showing that traditional psychoanalytical oriented psychotherapy is more effective than placebo plus psychotherapy (Daneman, 1961). One study exists which provides tentative support for the superiority of combined drug therapy/psychotherapy intervention over drug therapy and psychotherapy alone in reducing symptoms (Weissman et al., 1979). Finally, it appears that marital therapy plus lithium may be more efficacious

than lithium alone in preventing marital failure in bipolar patients (Davenport et al., 1977).

Overall, traditional psychosocial approaches to the treatment of depression have fared poorly relative to pharmacological treatments (for excellent reviews see Whitehead, 1979; Weissman, 1978; Hollon and Beck, 1979) in reducing depressive symptoms.

C. BEHAVIOURAL THEORIES

1. Overview

This section covers the major behavioural models of depression, most of which are Skinnerian or operant derivatives. Ferster (1947), Lewinsohn (1974, 1975, 1976), Seligman (1976) and Rehm (1977) have been the major contributors, though others have put forward classical or affect-mediated (Hollon et al., 1979) theories which focus on the role of conditioned anxiety and avoidance behaviour in the development of depression (Wolpe et al., 1966; Wolpe, 1971). As there appears to have been little systematic testing of the latter, only operant approaches and supporting evidence will be considered here.

Environmentally-oriented behavioural models implicitly reject the mediational variables suggested by psychoanalytic theorists (retroflected anger, loss of self-esteem). Rather, they construe depression in terms of reduced contingencies of reinforcement (Ferster, 1965; Lazarus, 1968), inappropriate contingencies of reinforcement (Ullman and Krasner, 1969) or a combination of these (Lewinsohn and Lisbet, 1972).

Ferster (1965, 1966, 1973) was among the first to evaluate depressive behaviours within an operant framework, albeit more exclusively from a theoretical standpoint than a pragmatic, empirical stance. He defined the fundamental characteristic of depression

as a reduction in frequency of behaviours that are positively reinforced and, basing his theory on analogue research from the animal laboratory, specified three components that might contribute alone, or in combination, to the development of depression. First, the frequency of positively reinforced behaviour will decrease if the schedule of reinforcement becomes too thin, i.e. if the individual is rewarded infrequently. Lewinsohn et al. (1969) have referred to this as a 'prolonged extinction schedule'. Second, the presence of maladaptive anxiety (e.g. instilled by high rates of punishment) can depress the rate of adaptive behaviour. Third, unexpected changes in the environment can lower the frequency of adaptive behaviour, particularly if cueing stimuli for ongoing behaviours are suddenly depleted, e.g. through loss, separation, death, or even organic deficits following illness. Ferster argues that depressives might have difficulty coping with low rates of reinforcement because of a lack of social skills, a stance taken by other behavioural researchers (Patterson et al., 1969; Moss et al., 1972; Lewinsohn, 1974). These investigators have expanded this research by focussing on low rates of response contingent reinforcement and aversive control (punishment, avoidance, escape) as aetiological factors in depression.

Klerman (in Becker, 1977) has criticised Ferster's claim that depression is essentially a weakness or absence of adaptive behaviour. Why is there a break in the impact of premorbid and intramorbid positive reinforcers? Why do such things as sex, food and work that were previously rewarding appear to lose their powers? Klerman suggests that the loss of reinforcer effectiveness may be due to internal, biological events rather than environmental changes.

Costello agrees with Klerman's hypothesis that apparent loss of reinforcer potency has been neglected. He, too, suggests a biochemical or neurophysiological change process as an explanation of diminished reinforcer effectiveness, particularly in depressed patients with endogenous symptoms. For those depressions characterised more by cognitive and mood disturbances the impact of rewards may be reduced by a disruption of a chain of behaviour at either an overt or covert level.

Drawing upon research from animal conditioning experiments, Costello says that loss of a significant person or context that served as a cue or prompt for significant rewards reduces the desirability of the rewards, which in turn seriously disrupts complex behavioural responses. He argues, 'that we are dealing with a chain of behaviours is suggested by the absence in many instances of any obvious link between the specific loss and his general loss of interest' (P.244, in Becker, 1977 (P.85)).

This is a highly speculative position and there is as yet little empirical support for the theory (Seligman et al., 1976). It is, however, of considerable heuristic value because of its testability and represents a direct attempt by behaviourists to deal with the problem of apparent loss of reward effectiveness.

Although Lazarus (1968, 1974) agrees with Ferster that depression results from excessive activity requirements per reward, he emphasises that depressed individuals undertake tasks they fail to complete, thereby depriving themselves of positive reinforcement. Furthermore, depressed behaviour is either deliberately, or inadvertently, rewarded by other people (e.g. attention or sympathy), thereby maintaining even more self-defeating behaviour. Various researchers have under-

scored the importance of reinforcement of depressed behaviour from social support systems (Bonime , 1966; Lewinsohn et al., 1968; Liberman et al., 1971; Stuart, 1967).

Treatment strategies based upon these theories focus upon the person's environment as a means of changing depressive behaviour. The therapist actively intervenes to arrange contingencies that will reward adaptive behaviours and extinguish or punish depressive behaviours. These methods attempt to change the way a person's family or associates respond to his behaviour, thus increasing the likelihood that he will engage in non-depressive behaviours (Welcrum et al., 1976). There are two controlled studies using this approach; both employ single case designs with reversals.

Reisinger (1972) treated a young female inpatient diagnosed as 'anxiety-depression' within a token economy programme. Crying behaviour was selected as the target to be reduced in frequency. The woman was given a token each time she smiled (adaptive response) and was fined a token each time she cried; a modified ABAB design demonstrated that the tokens exerted control over these behaviours. Several weeks later social reinforcement for smiling was paired with tokens. Tokens were gradually faded from the programme until social reinforcement was the only reward. The programme seems to have been successful as the resident was discharged shortly thereafter and had not been rehospitalised at follow-up 14 months later.

The main difficulty with this study is the implicit assumption that depression is equivalent to crying behaviour; other aspects of symptomatology were not considered (e.g. mood). Also, the generalisability of the results to other patients is severely restricted by the highly circumscribed nature of the depressed behaviour.

Hersen et al. (1973) demonstrated that a token economy programme reduced observable depression with three neurotically depressed inpatients, all of whom also received drug therapy. The programme focussed on behaviours in several areas: work performance, grooming, occupational therapy and responsibility. Talking, smiling and motor behaviour were assessed,on a time sampling basis, by the nursing staff using a behaviour rating scale. ABA reversal designs were employed. During the baseline conditions obtaining tokens was contingent upon performance of the target behaviour but the tokens had no value (privileges were non-contingent); both the behavioural ratings and amount of earned tokens were low. During the treatment phase, privileges were contingent upon tokens, and there was a marked increase in the number of tokens earned and a related increase in the behavioural ratings. However, both the behavioural ratings (smiling etc.) and number of earned tokens decreased remarkably when the baseline conditions were reinstated. Thus, all of the patients improved during the active treatment, reverting to pre-treatment levels during the reversal phase.

The strength of this study is that patients responded to treatment in a very short time (several days) compared with the four months in the Reisinger study. Its basic drawback is that like the previous study, no measure of affective state was taken. Furthermore, the fact that the behaviour changes were under such rigorous stimulus control reduces the potential of generalisation of the learned responses to other settings.

Lewinsohn (1968, 1974, 1975, 1976), Seligman (1976) and Rehm (1977) have preferred to maintain closer links between hypotheses and data than the earlier behavioural researchers. The contributions

of these learning theorists to the understanding of depression will be considered separately.

2. Lewinsohn's theory

According to Lewinsohn's viewpoint, depression is caused by a low rate of response-contingent positive reinforcement, which translates as positive reinforcement that is only forthcoming if the critical behaviours are emitted by the person (Lewinsohn, 1974). This low rate exists if (1) few events are rewarding, (2) few rewarding events are available in the environment and/or (3) the person lacks the necessary social skills to make those responses that would be rewarded. Thus, Lewinsohn's model is one that claims that it is not reduced reinforcement per se but an appropriate rate of response-contingent positive reinforcement that is crucial to the development and maintenance of depression.

Supporting evidence for the theory has been largely correlational (e.g. Bunner, 1975; Lewinsohn and Libet, 1972; Lewinsohn and Graf, 1973; Lewinsohn and Macphillamy, 1974; Macphillamy and Lewinsohn, 1974; Sheslow and Erikson, 1975; for reviews see Blaney, 1977). Essentially, these studies report negative correlations between reported levels of pleasant events and mood but do not establish any causal link between events and affect. For example, Lewinsohn and Libet (1972) and Lewinsohn and Graf (1973) found a correspondence across time within person's between participation in pleasant activities and depression. Considering the usual definitions of depression, this result is hardly more than tautology. These researchers, arguing that aetiology would be suggested if a decrease in activities preceded decreases in self-reported mood level, computed time-lagged correlations to evaluate the

extent to which there was a powerful relationship between behavioural changes on one day and a congruent mood change on following days. Neither study reported data compatible with the predictions of the theory, i.e. they failed to show that changes in activity preceded changes in mood.

Lewinsohn argues that lack of social skills is a causal factor in depression, the point being that reduced social skills decreases the amount of reinforcement available to the individual. This contention is held to be supported by investigations which demonstrate that depressed patients elicit less reinforcement from others and seem to show deficits in social skills (Lewinsohn and Shaffer, 1971; Lewinsohn et al., 1970; Libet and Lewinsohn, 1973; Libet et al., 1973). Since these correlational studies were conducted using depressed populations, it is likely that the observed deficits in social skills behaviour are merely aspects, rather than causes, of depression. These data also fail to address the issue of whether social skills deficits remain after remission of symptoms. Furthermore, one study has shown that depressed individuals may be as socially skillful as nondepressed people when placed in an 'analogue interaction situation' (Schrader et al., 1978). These investigators suggested that, rather than having a depleted repertoire of social skills, depressives may respond to certain kinds of situations by inhibiting acquired skills. Such a view is compatible with findings cited earlier that depressives appear to selectively modulate assertive behaviours in response to perceived social status deficits (Weissman and Paykel, 1974; Weissman et al., 1971).

Blaney (1977) has provided an excellent review of the relevant literature. In his view, 'since so much of the effort surrounding

Lewinsohn's theory has been essentially correlational, perhaps the theory should be treated as a characterisation of the depressed person's interaction with his environment rather than as a hypothesis concerning the causal antecedents of the depressive episode'(P.210).

Despite these challenges, Lewinsohn's model has had a major impact on behavioural theory and practice. The guiding principle behind Lewinsohn's therapy is to restore an adequate schedule of positive reinforcement by altering the level, quality and range of the patient's activities and interactions. This is done through the judicious application of graded tasks, contingent reinforcement and activity schedules (Bonham, 1979).

Thus, Hammen and Glass (1975) attempted to reverse depression in a nonclinical population using some of the techniques recommended by Lewinsohn (also see Rush and Beck, 1979, for a description of Lewinsohn's method). On the basis of scores on questionnaires in the mild to moderate range of depression, college students were randomly allocated to one of four conditions: the experimental group was instructed to increase their activities from an individualised Pleasant Events Schedule; another group was told to eat more protein than carbohydrate foods - this was used as an expectancy control group; also included were self-monitor (recording daily activities) and waiting list control groups. Subjects were not told that they were having 'treatment'. Although the experimental and self-monitor groups both maintained a similar level of pleasant activities, subjects in the 'treatment' group showed less reduction of depression than subjects who simply recorded their activity. Moreover, subjects who increased their activities reported fewer highly pleasant activities (self-rated). Thus, the circumstances under which pleasant activities

are increased appear to affect the impact of increased activity on mood.

The results indicate that depressed people seem to be able to participate in more pleasant activities but this does not necessarily improve their mood. Lewinsohn argued that the investigation failed to establish whether the prescribed activities were potentially reinforcing for the subjects. The brief treatment period (two weeks) and the fact that the subjects did not realise they were receiving treatment might also account for the findings (Whitehead, 1979).

Operating on the idea that depression is a 'reaction to reinforcement loss', Shipley and Fazio (1973) selected student volunteers who complained of depression lasting at least three weeks (screened using Zung Self-Report Depression Questionnaire). They provided functional problem-solving advice in an individual approach directed towards appropriate modes of social interaction. Homework assignments were given. Twenty-two treatment subjects and twenty-eight controls, i.e. an interest support group without homework assignments, were seen for three one-hour sessions over three weeks by one therapist. The results showed significantly greater improvement in the behavioural group compared to the control groups.

There are two problems here, also evident in the study conducted by Hammen and Glass (1975). First, there is an implicit assumption that a depressed population has been accurately identified. Self-report methods of defining and identifying depressed subjects embodies many critical problems. These problems relate to issues concerning the use of rating scales as diagnostic measures, in general, as well as to specific difficulties inherent in self-rating scales (e.g. Zung, SDS, BDI, etc.), as opposed to observer rating scales and

research diagnostic interview schedules (Depue and Monroe, 1978). Secondly, the severity of symptomatology in the populations studied places powerful constraints on the generalizability of the results to patients with greater levels of mood disturbance. With regard to Shipley and Fazio's work, Whitehead (1979) says, 'comparing the initial scores on the Zung SDS with those quoted for patients by Bigger et al. (1978), these student subjects scored in the range of nil to mild depression' (P.500)

The objective of improving social skills has been approached using a group modality. Therapy focusses on the acquisition of communication skills through graded task exercises and the discussion of specific interpersonal problems (Lewinsohn, 1970). The relationship between loss of reinforcement and depression is explained in some detail to provide a rationale for therapy. Although no controlled trials exist, in an investigation to be discussed in the next chapter, Shaw (1977) employed a similar procedure. He found that in college students seeking help for depression, the social skills group performed no better than subjects in a non-directive therapy control group.

In summary, Lewinsohn has developed a major line of behavioural research focussing on the importance of low rates of response contingent positive reinforcement as a key factor in depression. While the model is built on an empirical base, much of the evidence held to be in support of the theory is equivocal; some studies (e.g. Hammen and Glass, 1975) raise challenges to Lewinsohn's theory. Nevertheless, it is notable that treatments which focus on behavioural targets have been effective in alleviating some forms of depression.

3. Learned helplessness theory

Seligman (1972, 1973, 1975), Miller and Seligman (1973), Seligman

et al. (1976) have proposed a theory of depression in which the concept of loss of reinforcement is a tertiary rather than a central component. His model acknowledges the extent to which mediational variables, i.e. cognitive set, may interact with specific environmental events in the development of reactive depression. The basic tenet of the original model asserts that lack of contingency between behaviour and reinforcement results in a set, or perception, of learned helplessness.

In earlier work, Seligman and his colleagues observed that after dogs had been exposed to several trials of inescapable electric shock by being restrained in a harness, they seemed unable to learn to escape shock when the escape response was permitted (Seligman and Maier, 1967; Seligman and Groves, 1970). Moreover, they noted that the dogs were able to learn the appropriate response only after they had been physically hauled into the shock-free part of the shuttlebox over many consecutive trials. Seligman suggested that the animal's escape responses had been extinguished in the inescapable shock trials and described this phenomenon as learned helplessness.

Learned helplessness, then, describes a behavioural state characterised by the non-emission of adaptive behaviours because the organism recognises no relationship between its own responses and relief from aversive events.

Seligman has argued that learned helplessness is a model for 'reactive depression'. Behavioural similarities between animals subjected to inescapable shock and depressed patients, in Seligman's view, include reduced levels of activity and aggression, sexuality, sociability and retardation; and both remit spontaneously. He cites parallels between the aetiology of learned helplessness and the aetiology

ascribed by psychodynamic authors to depression such as Bibring (1953). Loss of appetite, and weight and brain depletion of a biogenic amine (norepinephrine), which acts as a neural transmitter, are also regarded as correlates between learned helplessness and depression (Becker, 1977). Seligman further suggests that depressed patients and laboratory animals benefit from antidepressant medications which increase the amount of centrally available norepinephrine. With regard to behavioural treatment, the animals have to be forced out of their helpless state by being dragged into a shock-free zone.

The cornerstone of Seligman's model is that depressed people believe that they lack control over environmental events. This cognitive set may result from a learning history which has not afforded an opportunity for positive control over the environment. For example, a history of failure in coping adequately with stressful situations, or recurrent punishment, may produce a negative set of expectations about one's ability to cope with future stressors. When confronted with stressful events such as reinforcement loss, people vulnerable to depression may give up easily in their attempts to problem solve, even though appropriate coping methods are available. Another example of a defective history would be if the person was rewarded regularly for behaviour but on a noncontingent basis. Under these conditions, the person might not learn the critical behaviours that are needed to elicit reinforcement from the environment (Wilcoxin et al., 1976), thereby generating a belief that the amount of incoming reinforcement has nothing to do with his behaviour. According to Seligman, this might account for 'success depressions' in which people attain a goal for which they have worked hard and feel that they are rewarded not for current behaviour but for who they are or what they have achieved

previously.

A number of human studies have been conducted both to induce states of learned helplessness and to demonstrate its existence in depressed subjects. Heroto (1974, 1975) showed that helplessness could be induced using aversive noise levels. It has also been shown that non-contingent positive reinforcement undermines competitive responding in students presented with discrimination problems (Kurlander et al., 1974).

Miller and Seligman (1973) investigated the effects of levels of depression and perceived externality of control on expectancy changes in skill versus chance performance tasks. Depressed college students were selected on the basis of scores on the Beck Depression Inventory (the mean scores of the highly depressed group were mild compared to clinical samples) and Rotter's Internal Versus External Expectancy Scales. In this study, expectancy change did not correlate with internal-external scores. Depressed and non depressed subjects did not differ on the chance task. However, the skill task, in which the reinforcement received was response-contingent, produced highly significant changes, i.e. depressed subjects perceived that they had less control over the amount of reinforcement they received than did non-depressed subjects working on the same task.

Although Seligman has not proposed any specific treatment strategies, he has referred to the approach used by Wadsworth and Barker (1977) as having a large element of 'control'. Their method, considered as an Antidepressive Program (ADP), seeks first to provoke, then to reinforce the expression of anger. They selected fifty six male inpatient depressives diagnosed as neurotic and psychotic, and used the Zung Self-Rating Depression Scale before treatment and there-

after at one and three weeks. Psychiatric patients received anti-psychotic drugs in addition to the other treatments. The experimental treatment consisted of making the patient perform repetitive, meaningless tasks until such time as he expressed anger and refused to do any more; at this point he was entered into group therapy. This was compared with an approach that used meaningful tasks, admittance into group therapy and pharmacotherapy (imipramine). The results showed no differences in response in the psychiatric patients, but for the neurotics, significant improvement was found in the experimental group.

It should be noted that this programme is linked to psychodynamic formulations in terms of the hypothesised role of anger in the treatment approach and might therefore be regarded as evidence in support of traditional psychotherapy. Methodologically, it might be argued that the results are an artefact of differences in patient admission since the experimental group received treatment before the control group. Also, the use of only a single depression measure makes it difficult to evaluate the magnitude of change in clinical terms. However, the result is at least arguable within the Seligman model insofar as patients in the experimental group were 'forced' in the initial phase to emit a response that was apparently incompatible with passivity (helplessness).

Several investigations (Gotchel et al., 1975; Klein et al., 1976; Roth and Kubal, 1975) have pointed to the role of increased anxiety and/or hostility in the helplessness paradigm. Drawing upon this evidence Blaney (1977) contends that the helplessness model may not be specific to depression. In addition, Rush and Beck (1979) note that 'the usual helplessness-inducing manipulations may also decrease self-esteem. This makes interpretation of many studies said

to support the helplessness model difficult, since self-esteem and helplessness may be confounded' (P. 307).

Extension of the learned helplessness model to clinical depressive disorders has brought strenuous criticism (for a complete review see Depue and Monroe, 1978). First, Seligman claims that the model is analogous to 'reactive depression'. Apart from the semantic confusion which arises from using the terms endogenous-reactive (neurotic) and psychotic-neurotic interchangeably, many investigations have shown a tremendous degree of heterogeneity inherent in the reactive group of patients (Kendell, 1976). According to Depue and Monroe 'it has become impossible to speak meaningfully of reactive depression as a unified entity or type or to speak of this group confidently within the framework of a single aetiologic factor' (P.7). Seligman's contention that learned helplessness is comparable to 'reactive depression' virtually discounts the confusion in this area and implies a distinct position on aetiological factors, i.e. depressive reactions are induced by environmental events.

Several of the symptom parallels between learned helplessness and clinical depression put forward by Seligman may be inaccurate. As in the case of reactive depressions, it is no longer possible to provide a simple definition for the so-called major depressions (e.g. unipolar and bipolar) because the clinical manifestations within each are so variable (Spitzer, 1975; Akiskal and McKinney, 1975). In viewing passivity, psychomotor retardation, and stupor as parallels between his model and reactive depression, Seligman appears not to have considered the relevant literature on symptomatology and depressive subtypes. These symptoms are not necessarily characteristic of depressive disorders in general, let alone reactive depression. Depue

and Monroe note that these features are more compatible with some forms of endogenous depression, bipolar 1 particularly, as measured by 24 basic behavioural ratings.

Finally, Seligman's empirical support rests heavily on human analogue studies of depression. While analogue studies can be helpful in delineating specific processes in depression, the use of self-report scales as diagnostic (selection) instruments (e.g. BDI), and the relatively low levels of mood disturbance in the populations studied makes it difficult to generalise the results to clinical samples.

Having recognised that the learned helplessness model is in need of revision with respect to these problems, the model appears to be worthy of continued investigation. It has a reasonably broad empirical base, good research generating potential, and may well have important implications for greater understanding of 'normal', situational and/or minor depressive disorders.

Recently, Abramson et al. (1978) have proposed a reformulation of the learned helplessness model which is said to overcome many of the shortcomings inherent in Seligman's original theory. Arguing on strictly academic grounds, these authors point out that the original model, when applied to humans, has two major drawbacks. These may be summarised in a quote from the paper.

> '(a) it does not distinguish between cases in which outcomes are uncontrollable for all people and cases in which they are uncontrollable only for some people (universal versus personal helplessness), and (b) it does not explain when helplessness is general and when specific, or when chronic and when acute' (P.49).

The revised model is based on a revision of attribution theory (Winer, 1978) and attempts to address these critical issues. It can

be summarised as follows:

(1) Depression is comprised of four kinds of deficits: motivational, cognitive, self-esteem and affective.

(2) Depression occurs when a person believes that he is unlikely to achieve a desired goal and expects that he lacks the coping responses necessary to increase the likelihood of reaching the goal (helplessness).

(3) If the person believes that he is helpless to respond to a broad range of situations or problems, then he is likely to experience deficits in a wider range of situations (generality). The narrower the range of situations in which the person believes himself to be helpless, the more situation-dependent the depression will be (specificity).

(4) Chronicity will depend on whether the state of helplessness is recurrent or long-lived. The more stable the state of helplessness is, the longer the duration of symptoms.

(5) Loss of self esteem will depend on whether the person blames himself (externality) for his helpless state.

(6) Severity of depression depends on the extent to which the person expects that he cannot control events. The more certain he is that he cannot exert positive control over his environment, the more severe the deficits.

In a word, the reformulated learned helplessness model says that once people perceive noncontingency between their responses and reinforcement, they then attribute their helplessness to a cause.

Of the formulations discussed thus far, the revised learned helplessness model of depression is the most recent and consequently has received the least amount of research attention. Although there

is no direct empirical support as yet, the authors argue that the reformulated hypothesis is consistent with the experimental evidence on learned helplessness in humans and findings from human analogue studies on attributions (see Abramson et al., 1978).

Abramson et al. have proposed a broad-based, but as yet untested, treatment strategy on the basis of the reformulated model. Some of the behavioural treatments discussed in the previous section may be useful such as graded tasks - providing realistic goals and standards of success as well as social and problem-solving skills training, whereby the individual learns a wider range of coping responses. They also suggest 'environmental manipulation by social agencies to remove aversive outcomes or provide desired outcomes, e.g. rehousing, job placement, financial assistance' (P.69). Another strategy involves the use of verbal-persuasive techniques in changing (1) the expectation from uncontrollability to one of controllability over circumstances, and (2) unrealistic attributions of failure.

In contrast to behavioural models which view therapeutic procedures in terms of establishing appropriate schedules of reinforcement, learned helplessness therapy focusses on changing two mediational or cognitive elements, (1) the belief that one cannot exert positive control over reinforcement, and (2) the belief that one is personally responsible for a wide range of aversive outcomes.

4. Self-Reinforcement Theory

Rehm (1977) has proposed a self-reinforcement theory of depression based on deficits in self-control. It incorporates features from the behavioural and mediational models of the disorder. Like the learned helplessness model, it acknowledges the centrality of thinking processes

in the aetiology and treatment of depression. On the other hand, self-reinforcement theory is in agreement with behavioural models in stating that response-contingent positive reinforcement is crucial in the maintenance of adaptive behaviours. The two models differ in that the behavioural stance holds that depression is caused by environmentally supplied reinforcers, whereas the self-reinforcement theory emphasises the rewards and punishments that the person delivers to himself.

The self-control theory states that depression is a consequence of defective self-reward mechanisms. The model takes its lead from proposals by Bandura (1971) and Kanfer (1970, 1971) that self-reinforcement consists of three components: self-monitoring, self-evaluation and self-reinforcement.

Self-monitoring refers to observing one's own behaviour and the events which precede and follow it. The term 'behaviour' is used in its broadest sense to include not only motor responses but internal events such as mood, proprioception and cognitive activity (e.g. selective attention, discrimination). Self-evaluation is the process of making judgements about one's performance, and estimates about the causes of positive or negative performance, relative to internalised standards. Self-reinforcement refers to the self-administration of appropriate schedules of positive reinforcement which may be regarded as a way of supplementing environmentally dispensed schedules of reinforcement.

According to the self-control theory (Rehm, 1977), depression is seen as a series of deficits in self-monitoring, self-evaluation and self-reinforcement. This tripartite self-control system may be defective at any of six substations in the behavioural chain. These

are (1) selective attention to negative events, (2) selective attention to immediate instead of delayed consequences of behaviour, (3) imprecise attributions of causality, e.g. attributing successful performance to chance as opposed to one's skills, (4) excessively high performance standards, (5) low rates of self-reward and (6) high rates of self-punishment.

People who are vulnerable to depression are said to have a learning history mainly characterised by low rates of self-administered reward and high rates of self-punishment. Bandura (1971) suggests several ways a person may develop faulty self-reinforcement systems with respect to certain behaviours. An individual might fail to self-reinforce if (a) he does not regard a problem as challenging his abilities, (b) he undervalues the importance of a task and (c) he has unrealistic expectations concerning his ability to solve the problem at hand. Bandura (1977) and Thoreson and Mahoney (1974) have reviewed a sizeable literature on the means by which performance standards are learned.

Excessively high performance standards may be one reason why an individual may self-reinforce infrequently, thereby leav ing him vulnerable to depression. Since it is very unlikely that an individual with such standards could generate behaviours that would match his expectancies, the resulting failure to live up to his aspirations produces a negative self-evaluation of performance. Rehm hypothesises that 'super-criteria' for self-reward can generate feelings of worthlessness, low motivation and depressed affect.

With regard to the presumed mechanisms that maintain established patterns of self-reinforcement and self-punishment, Wilcoxin et al. (1976) state:

'self-reinforcement is maintained by periodic social reinforcement. The performance of punishable behaviour is hypothesised to generate anxiety or guilt. Self-punishment terminates these distressing thoughts and forestalls possible social condemnation. In addition, self-punishment may elicit social approval from others who are pleased with a person's efforts to control his own behaviour' (P.220).

The self-control model has empirical support. For instance, there is evidence that depressed populations demonstrate an attentional bias to negative events (Loeb et al., 1964; Loeb et al., 1971; Weintraub et al., 1974; Hammen et al., 1976). It has been shown that depressed subjects underestimate the amount of positive reinforcement they receive (Wener and Rehm, 1975). However, this literature is also compatible within a cognitive analysis (Beck, 1976). In a test of a hypothesis derived from Lewinsohn's theory, Rehm and Plakosh (1975) reported correlational data supporting the view that depression is related to a preference for immediate rather than delayed reward. Two unpublished studies mentioned in reviews by Hollon and Beck (1979) and Rush and Beck (1979) suggest that depressed individuals dispense self-punishment more often than non-depressed subjects, whereas depressed subjects deliver less reward (Roth et al., 1974; Rehm, 1977). Rehm also cites evidence which indicates that attributions of causality vary as a function of observed covariance between events (Kelly, 1971) and studies which suggest that accurate attributions of responsibility mediate changes in cognitive and behavioural responses, e.g. the learned helplessness work of Klein and Seligman (1976).

Rehm (1977) has devised a therapy package which consists of different behaviour therapy techniques that focus on each of the separate self-control deficits. Essentially, self-control therapy involves

training depressed individuals to attend selectively to and record positive events. Self-monitoring diaries are used as a basis for discussion during therapy sessions in which the therapist attempts to modify the individual's distortions of their own behaviour. This skills training package also includes teaching people how to monitor long-term as well as short-term consequences of behaviour, the direct modification of self-attributional deficits, assisting individuals to evaluate their performance realistically by making criteria for success less stringent, and the use of appropriate self-reinforcement techniques.

A behavioural programme for depression based on the self-control model has been developed and tested in a controlled treatment trial. Fuchs and Rehm (1977) treated volunteer depressed subjects who were screened using the Minnesota Multiphase Personality Inventory (MMPI) in weekly group sessions over a six-week period. The self-control programmes consisted of didactic presentations of self-control concepts and homework assignments focusing on appropriate self-evaluation and self-reinforcement (N = 8). This approach was compared with a non-specific treatment (N = 10) which involved discussion of problems, social interaction, and self-disclosure but no behavioural assignments were given. Compared to a waiting list control (N = 10), both treatment groups showed improvement on self-reported mood (BDI) and the self-control programme demonstrated significantly greater improvement than the nonspecific group at the end of treatment. A six-week follow-up assessmemt indicated a continuing but nonsignificant trend for the superiority of the self-control treatment over non-specific therapy.

The findings suggest that treatment based on a self-control model is more effective than nonspecific and waiting list controls. However, these depressed subjects cannot be regarded as a clinical

sample - those with a history of psychiatric hospitalisation or suicidal ideas or attempts were excluded from the study. Depressed volunteers may respond differently to treatment than an identified psychiatric population.

In terms of the theory, the self-control model is open to many of the criticisms that have been directed towards the behavioural and learned helplessness models, e.g. heavy reliance on correlational and human analogue findings as principal sources of evidence. However, like these models, Rehm's theory is phrased in operational terms, has an empirical base, and as such, remains one of several psychological theories of depression that can be scientifically tested.

While the self control model has been associated with operant behavioural theories, its distinct emphasis on the role of covert processes in the aetiology and treatment of depression (e.g. selective attention, attributional style) places it firmly in the camp of mediational, or cognitive, paradigms. As will be seen, Rehm's concept of self control deficits bears a striking resemblance to Beck's cognitive theory and therapy of depression (Beck, 1976).

From a mediational viewpoint, the major drawback of the environmentally oriented behavioural theories of depression (Ferster, 1973; Lazarus, 1968, 1974; Lewinsohn, 1974, 1975, 1976) is that they present an impoverished outlook of a highly complex psychobiological process. In assuming that depression is primarily a behavioural disorder, they appear to discount the wide variety of clinical problems encountered in the psychiatric clinic. Moreover, environmental models neglect the role of thinking and individual differences in information processing and their relationship to mood, processes that appear to have major implications for theory and practice (Hollon and Beck, 1979).

This section reviewed the main contributions of behavioural approaches to the field of depression. While the behavioural tradition flatly rejects the intrapsychic model favoured by many psychodynamic theorists, they have retained links with the analytically derived but testable concept of loss. From a behavioural perspective, depression is seen as a consequence of maladaptive interactions with the environment which relate to several aetiological hypotheses. These include (1) loss of reinforcement, (2) loss of reinforcer effectiveness, (3) inappropriate schedules of reinforcement together with a lack, or loss, of social skills, (4) perceived lack of control over reinforcement, and (5) self-control deficits.

Historically, the emphasis has shifted from the influence of external events, i.e. environmentally dispensed reinforcement, on overt behaviour towards the role of covert processes (e.g. selective attention) in the development and maintenance of depression. It is worth noting that the mediational theories, in particular learned helplessness, have aetiological features similar to those put forward by neoanalytic writers such as Bibring.

Treatment approaches based on behavioural models appear promising in view of the demonstrated efficacy of these methods on subclinical depressed subjects. However, these findings must be confirmed in replication studies and it remains to be seen whether behavioural strategies will be effective in the treatment of more serious depressive disorders.

Table $3C_1$ summarises the psychoanalytic models which have been reviewed. Table $3C_2$ outlines the behavioural theories offered to date and Table $3C_3$ shows the behavioural models which incorporate important cognitive elements. As seen, the distinction between traditional behavioural and mediational behavioural models is becoming less defined.

Table $3C_1$ Psychoanalytic Models of Depression

Authors	Theory
Abraham (1911, 1916) Freud (1917)	Anger directed against the self following real or symbolic loss
Rado (1928)	Rebellion of the ego against a cruel superego, e.g. struggle to regain superego's approval through self-criticism
Fenichel (1945)	Unconscious aggression felt for lost object is discharged onto the ego, 'struggle to maintain self-esteem'.
Bibring (1953)	Collapse of self-esteem, person cannot live up to his ego ideals and becomes aware of his helplessness to attain them.
Jacobsen (1953)	Poor development of early self, and object representations lead to blurring of boundaries between the ego and ego ideals, hence maladaptive self-appraisal mechanisms.
Bowlby (1960)	Separation from mother at early age, loss of loved object in childhood increases vulnerability.

Table $3C_2$ Behavioural Models of Depression

Authors	Theory
Ferster (1965, 1973, 1974)	1. Loss of principal reinforcer leads to reduced positive reinforcement for adaptive behaviour.
	2. Social skills deficit leads to impaired ability to recover from reduced positive reinforcement.
Lazarus (1968, 1974)	1. Loss of reinforcer, acting as a discriminative cue, leads to broken chains of behaviour.
	2. Deprivation of reinforcement by failing to complete tasks.
	3. Social system supplies reinforcement for depressive behaviour.
Lewinsohn (1968, 1974, 1975, 1976)	1. Low rate of response - contingent positive reinforcement.
	2. Few events are reinforcing.
	3. Few reinforcing events are available in the environment.
	4. Social skills deficit (less frequent and skillful and more non-contingent reinforcement from others)

Table $3C_3$ Behavioural Models of Depression which incorporate a mediational (cognitive) component

Authors	Theory
Costello (1974)	Loss of reinforcer effectiveness leads to overall disruption of chains of behaviour.
Seligman (1972, 1973, 1975)	Perception of the lack of contingency between behaviour and reinforcement leads to a state of 'learned helplessness' (a belief that one lacks control over environmental events)
Abramson, Seligman, Teasdale (1978)	1. Belief that one lacks control (helplessness) 2. The person then attributes his/her helplessness to a cause. 3. Internalised, global, stable attributions lead to generalised and prolonged helplessness
Bandura (1971) and Kanfer (1970, 1971)	Reduction in self-reinforcing thoughts and activities
Rehm (1977)	1. Selective attention to negative events. 2. Selective attention to immediate instead of delayed consequences of behaviour. 3. Inaccurate attributions of causality. 4. Excessively stringent performance standards. 5. Low rates of self reward. 6. High rates of self-punishment. 7. These are essentially deficits in self-monitoring, self-evaluation and self-reinforcement.

D. BROWN'S SOCIOLOGICAL APPROACH

While the psychodynamic and behavioural approaches differ in several important ways, they are alike in terms of their emphasis on the individual as a means of investigating aetiological factors in depression. This clinical or intensive approach to aetiology finds its origins in the history of medicine (Zilboorg and Henry, 1949). Detailed knowledge of the person and his disorder allows the investigator to make sense of the meaning of the person's symptoms, predict the course of the disorder and, if possible, plan remedial action to correct the problem. This is the main advantage of the clinical approach. The trouble is that such detail makes it difficult to progress beyond the individual case (Brown and Harris, 1979).

Another way of conducting aetiological research in psychiatry is the epidemiological survey in which all cases of psychiatric disorder in a population sample are ascertained. Selective information is obtained both in cases and in normals. This has the strength of representative sampling to avoid the potential bias in examining treated cases, i.e. that it may only be treatment seeking behaviour that is being investigated (Paykel and Rowan, 1979). Against this, the epidemiological survey maintains comparability at the expense of ignoring much of the complexity and clinical richness of the individual case.

Brown and Harris (1978), in their controversial book 'The Social Origins of Depression', argue for the compatibility of the clinical and epidemiological approaches in aetiological research. In their view, a sociological model can make two important contributions to the field of depression. First, it can investigate whether factors in the social environment, e.g. enforced housing, can increase the probability of developing a psychiatric disorder. Secondly it can assess how the

individual perceives and reacts emotionally to these conditions and other life events.

Since the work of Brown and his colleagues is closely related to research on life events and depression, studies on this topic are considered here in a separate section.

1. Life events

Investigations have established the relationship of life events to general illness (Holmes and Masuda, 1974; Rahe, 1974; Wyler et al., 1971) and psychiatric disturbance in community samples (Myers et al., 1974; Brown, 1973, 1975, 1978). A number of studies have also compared events reported prior to onset by depressed patients with those reported by control groups.

Paykel et al. (1974) looked at life events in 185 depressed inpatients and a similar number of matched controls from the general population. Life events changes during the six months prior to onset of symptoms were measured by interview with patients some weeks after symptomatic improvement. The depressed patients had three times as many life events changes as control subjects. When events were divided into 'desirable' and 'undesirable' categories, depressives were found to report significantly more undesirable events but did not differ from the controls on desirable events. Paykel also devised a measure of exits (departure of someone from the immediate social field of the respondent) and entrances (introduction of an individual). Depressives had a significantly higher number of exit events compared with controls, though there was no excess for entrances.

Comparing depressed women with a sample of women from the general population, Brown et al. (1973) found that the rate for all

events was raised only in the three weeks preceding onset of depression, but for events rated as very threatening (on a combination observer and personal response measure) it was elevated over the full 48 weeks studied. In a smaller study, Thomson and Hendrie (1972) found that depressives scored higher on weighted stress measures than hospital personnel. Consistent with psychoanalytic theories, Cadent et al. (1972) found that depressed patients reported twice as many recent losses as their relatives, though the difference did not attain a satisfactory level of significance.

Using depressed patients as their own control, Paykel (1974) compared life events at symptomatic onset with short-term follow up. Thirty patients from a randomly selected sample of seventy were found to have been symptom free for at least six months. These thirty subjects had approximately half the percentages of exits and undesirable events of the initial sample. While events rates fell, they were still higher than general population levels.

There is evidence linking life events to other specific psychiatric disorders. For instance, Brown and Birley (1968) reported a significantly higher level of events in the three weeks prior to onset or relapse of schizophrenia than in the general population. High incidences of life events have been reported in schizophrenics relapsing on drug treatment in maintenance trials (Liff et al., 1972). Jacobs et al. (1976) found that first admission schizophrenics experienced more undesirable events in the year before symptom onset. These investigations suggest that schizophrenics may be particularly vulnerable to emotional interchanges with the environment (Brown et al., 1972).

Paykel (1974) cites a study by Jacobs in which fifty schizophrenics and fifty depressives were compared on life events. Significantly fewer

undesirable events, exits, and total life changes were noted in the schizophrenics compared to the depressives.

Beck and Worthin (1972) have also investigated life events in schizophrenic and depressive samples. They found that depressives reported more events and events of higher mean rated threat than schizophrenics.

Paykel (1974) investigated suicide attempters, compared with matched mixed depressives, and controls from the general population. Suicide attempters reported four times as many life changes as the controls and 50% more than the depressed patients. Attempters had significantly more undesirable events than the depressives and the general population. Threatening-type events differentiated the attempters from the depressives, but interpersonal function was a common occurrence before suicide attempts and onset of depression. In the month before suicide attempts, events were especially high (Paykel, 1979, in Paykel and Rowan, 1979).

Other studies have pointed to the role of life events in samples of neurotic patients attending general practitioners (Cooper, 1973) and mixed psychiatric and medical/surgical patients (Schess et al., 1977; Morrison et al., 1968).

Impressive as these retrospective studies may seem, they have a common methodological flaw. Having experienced illness may influence people to view their past experiences differently, whereby they identify more changes in life events than actually occurred (Parsons, 1975). Prospective studies, such as those of Holmes and Macuda (1974) and Rahe (1974) reduce the problem of retrospective bias and provide a better indication of predictability of illness.

In summary, these studies give strong support to the hypothesis that life events precede a wide array of psychiatric problems. Depressed

patients are more likely than controls to report more life events that are undesirable and involve loss of people from their social environment during the six months prior to onset of illness than are controls. There is evidence that schizophrenia bears some relation to life events, but perhaps less than depression.

2. The theory

In keeping with their philosophy of combining the clinical and epidemiological methods to research, Brown and Harris (1975, 1978) have proposed a provocative sociological theory of the origin of depression. They argue that it is the interaction of life events (provoking agents) with predisposing circumstances in the social environment (vulnerability factors) that generates depressive illness.

They compared 114 depressed females and an epidemiological community sample of 458 women. Information was obtained by interview and it was found that 8 per cent of the community sample had onset of psychiatric symptoms, primarily depression, within the previous year. These were designated as 'onset cases'. Severe life events and serious long-term problems were highly correlated with depression in both groups. However, the investigators were also concerned with predisposing factors, i.e. social factors which increased the likelihood of symptomatic onset after the occurrence of a life event. These vulnerability factors included: three or more young children present in the home, lack of a confidant with whom the person could share apprehensions, lack of employment, full or part-time, and loss of a mother by separation or death before age 11. These variables seemed to account for the similarly strong relationship shown by lower social class.

The data for predisposing factors emerged mainly from the community

population instead of the patients. Brown and Harris suggested that other treatment seeking factors prompted vulnerable individuals not to explore available sources of medical care. It was argued that caring for young children in the home makes it less likely that women will consult a general practitioner. Also, it may be that the different meanings that patients and less disturbed women attach to the onset of symptoms affects whether an individual will seek treatment. While these are plausible explanations, Paykel et al. (1979) point out that mild rather than clinical depression could account for the high prevalence of depression in the community sample.

Pursuing the line of deprivation researchers, Brown et al. (1977), looked at specific kinds of losses in childhood in relation to different types of depression. They found that neurotic depression was related to general losses, whereas loss by death was associated with psychotic depression. It was suggested that the more severe the loss, the greater the negative psychological impact (loss of self-esteem, increased sense of helplessness).

There is evidence for the relationship between lack of social supports and minor psychiatric disturbance. Miller and Ingham (1976) and Miller et al. (1976) found that lack of a confidant and friends appeared to both regulate the effects of life events and to be directly related to symptoms, suggesting an independent effect for vulnerability factors.

With regard to provoking agents, Brown and his colleagues hypothesise that both the time intervening between a given life event and its severity in terms of perceived threats are related to the onset of psychiatric illness (Brown et al., 1973). Moreover, various events happening to the same person, less obvious than bereavement or divorce, may interact

and lead to symptomatic onset. Miller and Ingham (1979) reported evidence of a small 'summation' effect for both major and minor stresses plotted against severity of depression, though a better predictor of symptom severity might be a dissipation model of life events (events stress dissipates gradually over time) as suggested by Surtees and Ingham (1980). Surtees and Ingham regarded their findings as compatible with the work of Brown and Harris: 'experiencing multiple discrete severe events results in a (small) increased risk of an onset of depression but . . . experiencing minor events, even in combination with severe events results in no additional risk' (P.29).

Shapiro (1979) has criticised Brown and Harris's work on the grounds that they were ' subjective' in their methodological approach with respect to patient selection, measurement of life events, choice of analyses, and interpretation of the data. They replied with a point-by-point defence of the methodology and argued that Shapiro's purist emphasis on experimental rigor , while understandable, is not easily accommodated within a multi-disciplinary approach to psychosocial research (Brown and Harris, 1979). Others have said that Brown's findings do not support the differentiation of risk variables into vulnerability factors and provoking agents (Tennant and Bebbington, 1978). However, the same authors reanalysed Brown's data and concluded that there appeared to be an effect for vulnerability factors in their own right.

While Brown's work is open to criticisms that can be made of many retrospective studies, it appears to be of seminal importance in the field of psychiatric aetiological research. An enormous amount of very detailed work has been done in order to establish that severe events in the life of a patient, particularly early loss, are among

the main causes of depression. As seen in this review, there is some evidence that this may be so. However, the most important aspect of Brown's research is its focus on identifying those people in the general population who are most likely to develop a clinical depression. Does exposure to vulnerability factors put an individual at greater risk to onset of depression? Clearly there is not enough information available yet to address this question adequately.

Ideally, a prospective study is called for, though this is much easier said than done. A compromise solution might be to attempt to replicate Brown's work with a view to establishing similar prevalence rates using more rigorous diagnostic criteria, as suggested by Paykel and Rowan (1979).

Brown and Harris stress that their model has implications for treatment. In their view:

> 'The results of this study suggest that the understanding of untreated depression can be of great help in the understanding of those that have reached the treatment setting, and thus that even severely disturbed inpatients might benefit if physical treatments were supplemented by what might be called social therapy designed to raise their sense of self-esteem and increase the alternative sources of value available to them in the long term . . . These findings provide backing for many reforms in our current social organisation, increases in the number of nursery school places and the number of part-time employment opportunities for women being some obvious candidates. They point, too, to the large areas of loneliness and isolation which exist amid our so-called affluence and to the important role they play in determining family health. To combat these and to build a sense

of mastery and self-esteem, which will render every member of the community more resilient to the buffets of experience, requires more than comforting talk in a surgery.' (P.292-293)

For those interested in the psychological treatment of depression a fascinating question must be: vulnerability factors notwithstanding, does the impact of life events upon the person depend upon his coping abilities and/or personality? While there is no direct experimental evidence linking personality, life change events and depression, clinicians and researchers in the area believe that it does (Wolff, 1961; Rahe, 1974; Hinkle, 1974). It has been suggested that people who remain free from illness possess 'psychological characteristics' which help them to cope with the impact of life events, even the more severe stressors. Among such attributes are an ability to shift relationships with abandon, self-protective concern over own well-being, an avoidance of responsibility (Hinkle, 1974 in Parsons, 1975). This is, however, little more than speculation and there is an urgent need for further work in this area.

Recent evidence on the efficacy of behavioural interventions on mild forms of depression (e.g.Shipley and Fazio, 1973; Fuchs and Rehm, 1977) raises the possibility that psychological techniques may serve to modify the impact of life changes upon the individual. Since the appraisal of threat appears to be as important as the actual aversive event itself, those approaches which aim to modify cognitive processes would seem to offer the most promise in relieving 'event-associated depressions'.

E. CONCLUSIONS

This chapter has been concerned with the major psychosocial theories of depression, the treatments derived from them and supporting evidence.

Psychoanalytic theories have been highly influential in the study

of the aetiology and treatment of depression. Because psychoanalytic concepts are phrased in metapsychological terms, it has been difficult to subject identifiable hypotheses to experimental validation. Nevertheless, analytic theories have been of heuristic value, suggesting hypothetical constructs such as deprivation and hostility for scientific research. Treatments for depression based on analytic approaches have fared poorly compared to pharmacological interventions.

Environmental theories of depression have an empirical base but appear to convey more about how depressed people interact with their environment than they do about the causal factors involved in the onset of depression. Behavioural models which incorporate a cognitive component offer a more sophisticated approach which attempts to delineate the role of thinking processes in relation to mood and motivational factors. Therapies derived from behavioural theories are more effective than 'expectancy' and waiting list control conditions in the treatment of some mild forms of depression. There is, however, no evidence that behavioural techniques work better than pharmacological treatments of depression.

Life events, possibly interacting with social factors, appear to be of outstanding importance in the study of the aetiology of depressive illness. Research is urgently required to delineate the role of coping abilities and/or personality in relation to life events and the impact they have on the individual.

This review indicates that a plethora of potential predisposing factors exists, including biological substrates in neurochemical, physiological and pharmacological functions, previous event and reinforcement histories enabling the development of coping skills, factors

of help-seeking affecting those who eventually get treatment, and personality variables leading to vulnerability to stress in general and specific events. Such considerations suggest a highly multifactorial aetiology.

Of the mediational psychological models put forward to date, Brown and Harris favour Beck's cognitive theory of depression, the subject of the next and final chapter of the literature review.

CHAPTER FOUR

COGNITIVE THEORY AND THERAPY OF DEPRESSION

A. INTRODUCTION

The previous chapter reviewed the major psychosocial approaches that have been put forward to explain and treat depression. It was argued that a markedly multifactorial aetiology is required to explain the various components of the depressive syndrome which may prove to be mutually interactive over time. As suggested by Akiskal and McKinney (1975) it may be possible to enter the system from any of several directions to trigger a full-blown clinical depression (e.g. physiological, psychological, social).

Behavioural theories have contributed significantly to increasing our understanding of depression, particularly with respect to how depressed people interact with their environment. The therapies derived from them have helped to pave the way for the operationalisation of psychotherapy, including procedural manuals (e.g. Beck, 1976; Rehm, 1977; Beck et al., 1978, and Beck et al., 1979) that describe the specific techniques and their sequence during the course of treatment (Weissman, 1979). Moreover, recent studies on the efficacy of behavioural treatments of depression (e.g. Shipley and Fazio, 1973; Fuchs and Rehm, 1977) suggest that intervention at the psychological level is one way to alleviate depression, though it may be that combined treatment approaches (i.e. drugs and psychological therapy) aimed at entering the system at several levels will prove to be more effective than either approach on its own (e.g. Weissman et al., 1979).

One of the clearest trends in contemporary psychology appears to be the merging popularity of cognitive perspectives (Weimer and Palermo, 1973; Dember, 1974). The impact of theory and research on central mediating processes and cognitive-symbolic mechanisms can be readily identified in many subsepcialties (Mahoney, 1977). Judging from the literature reviewed in the previous chapter, behavioural approaches to depression have followed suit (Seligman, 1976; Abramson et al., 1978; Rehm, 1977). The attention paid to cognitive factors in traditional behaviour therapy has also increased remarkably over the last ten years, while several recent developments have strengthened the trend towards a cognitive-mediational model of the modification of behaviour (Bandura, 1974, 1977; Wilson, 1978). For instance, there are already several books devoted to the area (e.g. Beck, 1976; Mahoney, 1974; Meichenbaum, 1977 and Foreyt and Rathjen, 1978), and two journals (Cognitive Therapy and Research and Biofeedback and Self-Regulation) serving to disseminate a growing literature.

Before discussing Beck's theory of depression, it would be helpful to consider briefly the historical development of the cognitive learning approach to behaviour change. Afterwards, the chapter is devoted to a detailed description of cognitive theory and supporting evidence and a comparison of this approach with alternative theories of depression.

B. HISTORICAL PERSPECTIVE

Maloney and Arnoff (1978) have provided an interesting historical account of what has been referred to as 'the cognitive-learning trend'.

They point out that 'such strange bedfellows as cognitive psychology and behaviour modification' (P.689) have been persuaded to cohabitate as a result of several major changes in the thinking of behaviouristic psychologists, clinical scientists from various disciplines and behaviour therapists in particular.

In the mid 1960's behaviourists became very interested in the phenomenon of self-control which was mirrored in the development of weight control programmes (Ferster et al., 1962; Stuart, 1967) and laboratory investigations of self-regulatory processes (Kanfer, 1970; Bandura, 1969, 1971). This in itself appears to have been a truly revolutionary occurrence in the field of behavioural research. According to Mahoney and Arnoff (1978):

> 'Prior to this, the prevalent and explicit assumption of behaviourists was one of environmental determinism (i.e. that the forces shaping a person's life lie primarily in the external environment). In fact the long-standing debate between behaviourists and humanists was basically focused on 'internalism' versus 'externalism' (Matson, 1973) . . . behaviouristic research on self-control also ushered in the acceptance of reciprocal determinism that emphasised complex and continuous causal interaction between the organism and its environment (Bandura, 1969, 1971; Thoreson and Mahoney, 1974). The human organism was no longer viewed as a passive product of environmental influence, but as an active participant in his or her own complex development' (P.690).

Another major shift in behaviouristic circles involved the adoption of the idea that human thoughts could be functionally analysed and altered by merely extrapolating the principles of behaviour change (Homme, 1965). In a position paper, Homme argued that 'coverants,

the operants of the mind' were basically observable events, provided that one acknowledges that they can be monitored by the person experiencing them. It was argued that mentalistic dualism, a stance so bitterly criticised by behaviourists, was certain to linger as long as strict environmentalists neglected the role of 'private events' in human performance. Homme went on to present a behavioural technology (coverant control) that involved specific tasks built around the cueing and reinforcement of salient thoughts.

These changes prompted a movement whereby behaviourists began to apply the principles of conditioning to covert processes such as covert sensitization, i.e. an aversion method based on visual images (Coutela, 1966, 1967). As Mahoney and Arnoff (1978) note, 'Covert conditioning constituted a significant development in the trend toward a cognitive-behavioural interface. For the first time in decades behaviourists were openly and actively scrutinising their subjects' self-reported thoughts, feelings and images. Not only were these formerly banished phenomena now welcomed as <u>targets</u> of clinical concern but they were also employed as a <u>means</u> towards inducing other behaviour change. Not surprisingly, when behaviourism made this tardy move into the black box, it brought with it the accoutrements of its stimulus-response tradition. The processes and procedures of laboratory conditioning were soon imposed on human thought processes and mediation came to be viewed as a chain of covert stimuli and responses'. In his classic text 'Cognition and Behaviour Modification', Mahoney (1974) argued convincingly against the conditioning model of cognition and proposed a revival (as far as behaviourists were concerned) of the mediational approach to human learning and performance.

Mahoney's erudite defence of the cognitive-learning model had been

anticipated by rumblings in the behaviour therapy camp as far back as the late 1960's. On the heels of the covert conditioning movement came Bandura's (1969) book 'Principles of Behaviour Modification' in which he proposed an explicit shift to cognitive and information-processing models of behaviour change. In this and a more recent work, 'Social Learning Theory', Bandura (1977) stresses the role of behavioural procedures in effective psychological therapies, but argues that the underlying processes of behaviour change involve cognitive-symbolic mechanisms. His contentions are supported by a sizeable literature ranging from awareness (Notterman et al., 1952; Dulany, 1962, 1968; Spielberger and De Nike, 1966) to vicarious learning in animals (e.g. Morgan, 1896; Crawford and Spence, 1939; Church, 1957; Corson, 1967) and in humans (O'Connor, 1969; Berger and Johansson, 1968; Bandura, 1971; Bandura and Rosenthal, 1966). Later, other behaviour therapists became convinced of the need to include cognition in a comprehensive model of behaviour change (e.g. Lazarus, 1971; Eates, 1971; Goldfried, 1971; Kanfer, 1971; Mischel, 1973).

While behavioural quarters were experiencing these growing pains, social psychologists such as Kelly (1955) and Rotter (1954) and clinicians such as Ellis (1962) and Beck (1963) made their presence felt in the cognitive-learning arena. Their research and theories, particularly their emphasis on cognition and the role of belief systems in emotional disorders, have prompted many workers in the area to regard them as the founding fathers of the popular cognitive-learning trend (Mahoney and Arnoff, 1978).

Albert Ellis has been one of the most persuasive writers in the field. He noted that several Greek and Roman philosophers, including Epictetus, as well as Buddhist thinkers perceived the close connection

among reason, emotion and behaviour and offered advice for changing behaviour by modifying thinking patterns (1962). His form of psychological counselling, Rational-Emotive Therapy (RET) has claimed a considerable professional following and enjoys much of the popularity of the more commercial 'thought management' schemes put forward by writers such as Coue (1922), Carnegie(1948) and Peale (1960). In contrast to 'positive thinking' approaches, Rational Emotive Therapy (RET) has gained a degree of scientific credibility as a procedure for the treatment of various neurotic conditions. For example, comparative outcome studies have compared RET with some form of behaviour therapy in the treatment of anxiety, particularly systematic desensitization and behaviour rehearsal, though several have compared RET with client-centred therapy (e.g. Di Loreto, 1971; Moes and Heinman, 1972; Meichenbaum et al., 1971; Meichenbaum, 1972; Kanter, 1975; Wein et al., 1975; Teigerman, 1975; Molesky and Tosi, 1976).

Thus, having confronted empirical challenges (see Mahoney, 1974) the original conditioning model of self control gave way to more mediational theories and behaviour therapists started to investigate the potential for social and cognitive psychology in the clinical field. The conventional, rigid structure of behaviour therapy began to be replaced by more flexible approaches to behaviour change. 'Broad-spectrum' behaviour modifiers began to talk about and test coping skills therapies that focused on training people how to solve problems more efficiently (D'Zurilla and Goldfried, 1971; Spevack and Shure, 1974; Mahoney, 1977; Meichenbaum, 1974, 1977). As seen in chapter three, animal experiments on learned helplessness merged into a cognitive theory of depression (Seligman, 1975). Indeed, the reformulated version of the learned helplessness model (Abramson et al., 1978) finds its roots

in attribution theory which has been of considerable influence in clinical conceptualisations (e.g. Bem 1970; Rotter et al., 1972; Lefcourt, 1976). Not surprisingly, the turbulent journey from a conditioning to a cognitive-learning base has resulted in a blurring of the boundaries between behaviour therapy and other conceptual approaches (Rainy, 1975).

As suggested, the 'cognitive revolution' has not been without its detractors (e.g. Greenspoon and Lamol, 1978; Ledwidge, 1978, 1979) and has stimulated numerous debates and arguments (Bandura, 1976; Catania, 1975; Stuart, 1972; Rachlin, 1974). For instance, Ledwidge (1978) has argued that cognitive behaviour modification is 'a step in the wrong direction' on the grounds that the approach represents a return to 'mentalism' and that cognitive and behaviour therapists use completely different techniques to bring about desired changes in behaviour. In a recent paper, 'Cognitive Behaviour Modification: Misconceptions and Premature Evacuation', Mahoney and Kazdin (1979) have challenged Ledwidge's assumptions about metaphysical dualism and the validity of classifying therapists according to the techniques they employ. As they note 'if any clear distinction can be drawn, the major difference between cognitive and less cognitive behaviour modifiers does not lie in their therapeutic procedures so much as in their rationale and selection of a given procedure in an individual case. The more cognitively oriented therapist is inclined to employ a behavioural procedure appropriate to the 'cognitive restructuring' presumed to be required' (P.1045). Moreover, as will become clear, cognitive theorists (and therapists) are firmly committed to the tenets and practicee of modern behavioural research, i.e. careful specification of treatment components, use of multiple outcome measures including behavioural assessment, etc.

Despite some criticisms, the merger of cognitive psychology and behaviour modification has been greeted with enthusiasm and intense research. However, the term 'cognitive-behaviour therapy' encompasses an admixture of diverse and often underdeveloped principles and procedures (Wilson, 1978). Recognising the many differences among proponents of this approach, Mahoney and Arnoff (1978) have nonetheless extracted the following similarities:

1. 'that humans develop adaptive and maladaptive behaviour and affective patterns via cognitive processes (e.g. selective attention, symbolic encoding, etc.)

2. these cognitive processes are functionally activated by procedures which are generally isomorphic with those of the human learning laboratory, and

3. the resultant task of the therapist is that of a diagnostician-educator who assesses maladaptive cognitive processes and subsequently arranges learning experiences which will alter cognitions and, in turn, the behaviour-affect patterns with which they correlate' (P. 692).

These authors emphasise that the cognitive-learning perspective has spawned the development of three major forms of cognitive therapies: problem-solving therapies (e.g. O'Zurilla and Goldfried, 1971; Mahoney, 1977), coping skills therapies (e.g. Kazdin, 1974; Goldfried, 1971; Suinn and Richardson, 1971) and the rational psychotherapies which include Rational Emotive Therapy (Ellis, 1962, 1970, 1973), Self-Instructional Training (Meichenbaum, 1973, 1977) and Cognitive Therapy (Beck, 1976, Beck et al., 1978, Beck et al., 1979).

In summary, the role of cognition in psychopathology has been dis-

cussed by many clinical scientists, but none more so than A.T. Beck and his colleagues at the University of Pennsylvania. Since the primary focus of this thesis is Cognitive Theory and Therapy of Depression, the discussion is now restricted to that topic.

C. COGNITIVE THEORY

The cognitive theory of depression upholds the view that it is psychological factors which are central to the understanding of depression and these are the targets at which therapy should be directed. The main proponent of this view is A.T. Beck who, like Ellis, was early struck by the pervasiveness of irrational thoughts and fantasies in neurotic disorders, particularly depression (Beck, 1963). His subsequent clinical research led him to postulate patterns of irrational cognitions that are common to various clinical syndromes (Beck, 1976). Processes such as selective attention, magnification and arbitrary (illogical) inference are believed to produce cognitive structures that vary in their general themes. Thus, for each of the neurotic disorders, Beck suggests different ideational contents (Beck, 1976).

The cognitive model posits three specific concepts to explain the origins of depression: (1) the negative cognitive triad, (2) schemas, and (3) systematic logical errors, or defective information processes.

1. The negative cognitive triad

The cognitive triad is a thinking pattern that leads the depressed individual to regard himself, his experiences, and his future in an unrealistically negative fashion.

The first ingredient of the triad centres around the person's negative view of himself. According to Beck *et al.* (1979), 'He sees himself as defective, inadequate, diseased, or deprived. He

tends to attribute his unpleasant experiences to a psychological, moral, or physical defect in himself. In his view, the patient believes that because of his presumed defects he is undesirable and worthless. He tends to underestimate or criticise himself because of them. Finally, he believes he lacks the attributes he considers essential to attain happiness and contentment' (P.11).

The second ingredient consists of the depressed person's tendency to construe his ongoing experiences in a distorted manner. The world is perceived as a place making exhorbitant demands on him, or presenting unsolvable problems which prevent him from obtaining cherished goals. He systematically misinterprets situations in a negative manner even though more reasonable explanations are available. The depressed patient regularly views his interactions with the world as indicative of defeat or deprivation.

The third aspect of the cognitive triad consists of a negative view of the future (hopelessness). Beck et al. (1979) explain: 'as the depressed person makes long range projections, he anticipates that his current difficulties or suffering will continue indefinitely. He expects unremitting hardship, frustration and deprivation. When he considers undertaking a specific task in the immediate future, he expects to fail.' (P.11)

The activation of the negative cognitive patterns is said to explain the other signs and symptoms of the depressive syndrome. Rush and Beck (1979) provide some specific illustrations: 'if the patient incorrectly thinks he is being rejected, he will react with the same affect that occurs with actual rejection. If he is pessimistic about the future and anticipates negative outcomes, he will feel low in energy, apathetic, and be reluctant

to initiate various tasks. Suicidal wishes result from a desire to escape what appears to be an unbearable situation or an insoluble problem. Seeing himself as inept, he overestimates the difficulty of normal tasks in life. He is indecisive because he believes any decision he might make will be wrong' (P.311-312)

The depressed person's tendency to rely excessively on other people is also explicable within a cognitive framework. The patient depends on help and reassurance from those around him because they appear to be far more competent and efficient than he believes himself to be.

Finally, even the classical vegetative symptoms such as 'psychomotor retardation', reduced positive motivation, and inertia can be seen as a direct consequence of hopelessness, or a pervasive sense of futility (Beck, 1976; Beck et al., 1979).

2. Schemas (rigid beliefs)

The second principal component of cognitive theory consists of the concept of schemas. This concept is used to address the question: What kind of mechanisms account for the negative, self defeating thoughts of the depressed patient?

Schemas consist of organised superordinate structures of attitudes, or beliefs (basic assumptions) which influence the way a stimulus is seen, filtered, processed and responded to. Beck et al. (1979) explain: 'any situation is composed of a plethora of stimuli. An individual selectively attends to specific stimuli, combines them in a pattern, and conceptualizes the situation. Although different persons may conceptualize the same situation in different ways, a particular person tends to be consistent

in his responses to similar types of events. Relatively stable cognitive patterns form the basis for the regularity of interpretations of a particular set of situations. The term 'schema' designates these stable cognitive patterns' (P.12)

Beck's concept of schemas is similar to that put forward by academic psychologists such as Bartlett (1932), Neisser (1976) and Miller, Galanter and Pribram (1960). According to Neisser:

> 'A schema is that portion of the entire perceptual cycle which is internal to the perceiver, modifiable by experience, and somehow specific to what is being perceived. The schema accepts information as it becomes available at sensory surfaces and is changed by that information; it directs movements and exploratory activities that make more information available, by which it is further modified.' (1976, P.54)

Neisser suggests that schemas function as information-accepting systems, much like a format in a computer-programming language. Formats specify that information must be of a certain sort if it is to be comprehensible. Other information will be ignored or will lead to meaningless results.

A schema also functions as a plan, of the type described by Miller, Galanter and Pribram. Neisser says:

> 'Perceptual schemata are plans for finding out about objects and events, for obtaining more information to fill in the format. One of their important functions in seeing is to direct exploratory movements of the head and eyes. But the schema determines what is perceived even where no overt movements occur (listening is a good example), because information can be

picked up only if there is a developing format ready to accept it. Information that does not fit such a format goes unused. Perception is inherently selective.' (ibid, P.55).

Blumenthal (1977) has commented that the identification of one's self, or the attainment of a self concept begins early in life and is surely one of the most basic schemas that an individual ever forms. Self preservation is meaningless unless the self is first delineated. According to Blumenthal,

'Should the developed self concept be radically changed or lost at any point in life, as sometimes happens, we witness the most extreme changes in human personality, including severe mental pathologies' (P.147-148).

Lewin (1935) proposed that the self concept may be a complex schema consisting of many integrated subsystems. These subsystems are subconcepts that are associated with different spheres of one's life but are nevertheless affected by the condition of the whole self system. Examples of such subconcepts are an individual's physical appearance, social role, and intellectual ability.

In Lewin's terms, Beckian schemas might be regarded as aspects of the self concept (character attributes) or a set of general rules that guide how a person reacts to specific situations. Such formulas determine how the person organises perceptions into cognitions (i.e. verbal or pictorial ideation), sets goals, how he evaluates and modifies his behaviour, and how he understands or copes with events in his life.

The most obvious types of rules are standards and regulations. (Beck (1976) says that the individual employs 'a kind of mental rule book to guide his actions and evaluate himself and others'

(P.42). The rule book becomes a yardstick against which he compares the 'rightness, or wrongness' of his own behaviour and that of others. Performance is also evaluated as successful or unsuccessful according to these rules. As Beck (ibid) notes, 'By drawing on these standards and principles, he instructs himself (or others) how to behave in a given situation. Afterwards, he can evaluate the feedback from his actions, make the necessary corrections and either praise or criticize himself for his performance' (P.42). Furthermore, these rules are said to assist the individual in the analyses of complex interpersonal situations. For example, when an individual is talking to someone, the listener will not only attempt to decode the message, but will extract a highly idiosyncratic meaning from the information given. The manner in which the listener will respond is determined by his personal assessment of the situation, e.g. 'was the speaker being rude, or humorous?' 'Should I retaliate or continue the conversation in an affable manner?'

The organisation of different experiences is determined by the kind of schema in operation at the time. Specific occurrences, such as stressful life events, can trigger schemas which have lain dormant for long periods of time. The kind of rule that is activated in a given situation determines how the individual responds. Beck et al. (1979) attempt to clarify this point:

> 'In psychopathological states such as depression, patients' conceptualizations of specific situations are distorted to fit the prepotent dysfunctional schemas. The orderly matching of an appropriate schema to a particular stimulus is upset by the

intrusion of these overly active idiosyncratic schemas. As these idiosyncratic schemas become more active, they are evoked by a wider range of stimuli which are less logically related to them. The patient loses much of his voluntary control over his thinking processes and is unable to invoke more appropriate schemas' (P.13).

Beck (1976) specifies some of the schemas, or basic assumptions, that might make an individual vulnerable to excessive depression or sadness. Some examples are listed below.

1. 'In order to be happy, I have to be successful in whatever I undertake'
2. 'To be happy, I must be accepted (liked, admired) by all people at all times'
3. 'If I make a mistake it means I'm inept'
4. 'My value as a person depends on what others think of me' (P.255)

Beck points out that such schemas can be related to what Karen Horney (1950) has referred to as the 'tyranny of the shoulds'. For example, he notes that once a person has the schema 'In order to be happy, I need to be loved by everybody', he is likely to supplement this with another belief 'I should make everybody love me'. Beck contends that the 'shoulds' and 'should nots' have a slave-driving quality and have much in common with Freud's idea of the superego. Other examples listed by Beck are:

1. 'I should be able to endure any hardship with equanimity'
2. 'I should know, understand, and foresee everything'
3. 'I should be the perfect lover, friend, parent, teacher,

student, spouse'

4. 'I should assert myself, I should never hurt anybody else'.

(From Beck, 1976, P.257)

Working independently of Beck, Ellis (1962) labelled such rules as 'irrational ideas'. However, Beck differs from Ellis in his assertion that schemas 'are generally not irrational, but are too absolute, broad and extreme; too highly personalized; and are used too arbitrarily to help the patient handle the exigencies of his life' (Beck, 1976, P.246). Thus, as far as Beck is concerned, maladaptive schemas differ from adaptive ones in terms of their inappropriateness, rigidity, and excessiveness (for a review of maladaptive schemas, see Kovacs and Beck, 1978).

As mentioned previously, the depressed patient gradually loses control over his cognitions, and it becomes increasingly difficult for him to use a more adaptive set of attitudinal guidelines to regulate his behaviour. The less severe the symptomatology, the easier it is for the depressed patient to regard his self-defeating thoughts with some degree of objectivity. However, once the depression becomes intense, the patient's thinking becomes increasingly dominated by negative cognitions, even though there may be no logical link between real-life events and his negative constructions about them. The role played by schemas in the depressive spiral is described as follows:

'As the prepotent idiosyncratic schemas lead to distortions of reality and consequently to systematic errors in the depressed person's thinking, he is less able to entertain the notion that his negative interpretations are erroneous. In the more severe states of depression, the patient's thinking

may become completely dominated by the idiosyncratic schema: he is completely preoccupied with perseverative, repetitive negative thoughts and may find it enormously difficult to concentrate on external stimuli (for example, reading or answering questions) or engage in voluntary mental activities (computations, problem-solving, recall). In such instances, we infer that the idiosyncratic cognitive organization has become autonomous. The depressive cognitive organization may become so independent of external stimulation that the individual is unresponsive to changes in his immediate environment' (Beck et al., 1979, P.13)

Beck explains the relation between thought and affect in terms of a feedback effect, such that cognitions stimulate congruent affects and vice versa. Once the negative cognitive triad has been triggered (by the superordinate schemas), Beck has identified a number of logical errors in the depressed patient's thinking. These comprise the third major component in the cognitive theory of depression.

3. Systematic logical errors

According to Beck (1967), faulty information processing accounts for a series of logical errors in the thinking of the depressed patient which strengthen and maintain the belief in the validity of his negative ideas, despite the availability of contradictory evidence. These cognitive distortions can be divided broadly into two main categories: response and stimulus sets (bias).

Systematic errors in response set include:

1. Arbitrary inference: the process of drawing a conclusion in the absence of evidence to support the conclusion or when the evidence is contrary to the conclusion, e.g. the depressed person, kept waiting by the therapist, thinks: 'he has deliberately left

in order to avoid seeing me', or a frown on the face of a passer-by may trigger the thought 'he finds me disgusting'.

2. Overgeneralisation: Drawing a general conclusion on the basis of a single incident, e.g. 'I can't do anything right', when only a minor isolated failure has been experienced.

3. Magnification and minimisation: errors in evaluating the significance or magnitude of an event that are so gross as to constitute a distortion, what Ellis has termed 'catastrophizing', e.g. some everyday difficulty will end up a disaster. This inexact labelling evokes an emotional response congruent with the descriptive labelling, not with the actual event.

4. Personalisation: a tendency to relate external events to oneself when there is no basis for doing so. This aspect of depressive thinking is related to other distortions, especially arbitrary inference. In psychotic states, the depressed patient may believe that he is the cause of all catastrophies in the world.

5. Dichotomous reasoning: the process whereby the depressed patient places all experiences in one or two opposite categories, e.g. good or bad, flawless or defective, immaculate or filthy. Usually the patient chooses the extreme negative to describe himself.

These logical errors in response set interact with another faulty cognitive process which Beck calls selective abstraction.

Selective abstraction is a stimulus set which consists of focusing on a detail taken out of context, ignoring the more salient features of the situation and concentrating on the whole

experience on the basis of this element, e.g. 'my friend ignored me, so he doesn't like me anymore', disregarding the fact that he may have been busy at the time.

Beck et al. (1979) point out that the attributes of the typical depressive thinking are analagous to those described by Piaget (1932/1960) in his descriptions of the thinking of children. They have provided some examples of the differences between what might be termed 'primitive thinking' and 'mature', or adaptive thinking, some of which are listed below:

1.	Nondimensional and global: 'I am fearful.'	Multidimensional: 'I am moderately fearful, quite generous, and fairly intelligent.'
2.	Absolutistic and moralistic: 'I am a despicable coward.'	Relativistic and non-judgemental: 'I am more fearful than most people I know.'
3.	Invariant: 'I always have been and always will be a coward.'	Variable: 'my fears vary from time to time and from situation to situation.'

(from Beck et al., 1979, P.15)

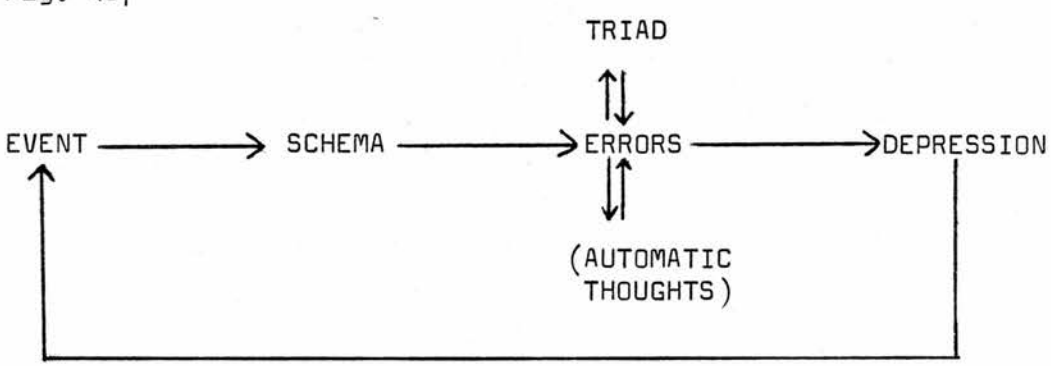
These examples suggest that depressed people may interpret their experiences in relatively primitive ways and tend to make wide-sweeping gudgements about events that impinge on their lives. As Beck et al. (1979) note, 'the meanings that flood their consciousness are likely to be extreme, negative, categorical, absolute and judgemental' (P.14).

In terms of what is seen in the psychiatric clinic, the

logical errors are most apparent in what Beck calls the depressed person's automatic thoughts, as they appear automatically and very quickly. Patients have to be taught how to monitor these thoughts (e.g. 'I'm weak', 'I'm a failure'), otherwise they may not even report them. As suggested earlier, these negative thoughts become more salient in severe depressive states. Automatic thoughts tend to be specific and discrete, autonomous, plausible, repetitive, and idiosyncratic within individuals and within disorders. They are essentially what other authors have referred to as internal dialogue (Meichenbaum, 1977) and self-talk (Ellis, 1962).

In summary, the cognitive model of depression puts forward three major ingredients to explain the basis of clinical depression: the negative cognitive triad, schemas, and systematic logical errors. Beck, therefore, considers thought processes to be the central factor in depression, all other signs and symptoms of depression being secondary to the maladaptive thought processes of the depression-prone individual. Figure $4C_1$ shows a diagram of the key components in Beck's model.

Fig. $4C_1$



4. Predisposing factors and precipitation to depression

The cognitive model offers an explanation as to how people become vulnerable to developing depression. Essentially, early

experiences provide the foundation for the development of the negative concepts of the self, the world, and the future. The depression prone person may become sensitised by stressful life events such as the loss of a parent or repeated rejection by peers. Other unfavourable experiences of a less outstanding nature may similarly produce vulnerability to depression. According to Beck (1976) 'these traumatic experiences predispose the person to overreact to analogous conditions later in life. He has a tendency to make extreme judgements when such situations occur. A loss is viewed as irrevocable; indifference, as total rejection.' (P.107-108)

For example, the break-up of a marriage may trigger the concept of irretrievable loss associated with the death of a parent in childhood. Sometimes the precipitating event is a physical illness or abnormality that activates the notion that the person is doomed to a life of unremitting agony. However, Beck emphasises that 'unpleasant - even extremely adverse - life situations do not necessarily produce a depression unless the person is particularly sensitive to the specific type of situation because of the nature of his cognitive organization' (Beck et al., 1979, P.16).

Other people who are at risk to developing depression set rigid, perfectionistic standards for themselves during childhood so that their expectations are dashed when they have to face inevitable disappointments in later life. Aberrant thinking is triggered in situations that impinge on specific vulnerabilities, such as acceptance-rejection, success-failure, health-sickness, or gain-loss (Beck, 1976). For example, a patient who believes that in order to be happy he must be accepted by all people at

all times will assess his own worth entirely by how often and to what extent he is accepted by others.

Beck suggests that these basic assumptions are derived during childhood from attitudes and opinions of peers or parents. At one time these beliefs may have been articulated and reflect basic family rules. For example, a parent might say to a child, 'Be nice all the time or Angus won't like you'. Conceivably, the child may repeat this aloud at first and later, at a subvocal level, to himself. After a while the child develops the underlying attitudes 'my worth depends on what others think of me'. Moreover, many of these attitudes are culturally reinforced (Beck et al., 1978).

While specific stresses (e.g. loss of job, disruption of a relationship) in conjunction with depressogenic schemes can precipitate adult depression, Beck contends that prolonged, non-specific stressors can also lead to a depressive episode. For instance, gradual withdrawal of affection by a partner or a persistent discrepancy between goals and achievements may also undermine the individual's psychological resources. Again Beck (1976) provides a clear illustration: 'The individual . . . may be continually dissatisfied with his or her performance as a parent, housewife, income producer, student or creative artist. Moreover, the repeated recognition of a gap between what a person expects and what he receives from an important interpersonal relationship, from his career, or from other activities, may topple him into a depression. In brief, the sense of loss may be the result of unrealistically high goals and grandiose expectations' (P.108).

Thus, depressed patients acquire their schemas as a result

of early learning experiences. The 'negative cognitive triad' may be grounded in experiences with disparaging parents or teachers or in a history of loss or repeated frustrations, with depressive affect being triggered by events that evoke negative cognitions. Such events tend to have cue properties similar to the early conditions that generated negative attitudes, though prolonged non-specific stress can also precipitate depression.

D. EVIDENCE FOR BECK'S MODEL

To evaluate Beck's model adequately, three points need to be demonstrated. First, the presumably causal elements (negative thoughts) must be shown to co-vary with the phenomenon under observation (depressed mood). In other words, that depressed patients think in a particularly negative manner relative to other populations. Second, it is necessary to demonstrate that changes in cognitive content and processes cause subsequent shifts in the other components of the depressive syndrome (affect, behaviour, motivation, and physiology). Third, it would lend weight to Beck's theory if it was demonstrated that the treatment derived from his theory was as effective or more effective than other methods of treatment of depression. This is not a strong test of Beck's theory, however, as the therapy he recommends contains many nonspecific factors such as attention and the manipulation of expectancies which are common to other types of therapy. Even so, it would be difficult to explain why treatments with a less cognitive approach were more effective. As this thesis is concerned with the effectiveness of Beck's therapy, special attention will be paid to this type of evidence.

The main sources of evidence for Beck's model of depression are

correlational, experimental, and treatment studies. These are reviewed separately.

1. Correlational studies

There is a large literature showing that depressive states have cognitive correlates (Beck, 1967, 1974). For example it has been demonstrated that negative perceptions of the self, the world, and the future are intercorrelated and that there is a relation over time between these negative cognitions and self-rated depressed mood (Weintraub et al., 1974). Minkoff et al. (1973) reported a high positive relationship between depression and hopelessness (General Expectancy Scale) and an even higher relationship between intent to commit suicide and hopelessness, though this relationship also held in a schizophrenic group. Katz et al. (1969 in Hollon and Beck, 1979) found that ratings of sadness were highly correlated with self-ratings of pessimism ($r = .56$) and of negative self-concept ($r = .79$) in a clinically depressed sample. Using self-esteem ratings to assess the self-evaluative aspect of depressive cognitions, Hammen and Krantz (1976) found that depressed women were significantly more self-critical than non-depressed women.

In a more recent article, Krantz and Hammen (1979) reported on a measure of cognitive distortion which reliably distinguished relatively depressed and non-depressed groups, including mild and clinical levels of depression. The instrument is said to assess the 'biased manner of evaluating situations that emphasize negative, self-critical, or pessimistic interpretations that are not warranted by the events themselves' (P.617). They found a consistent positive

relationship between depressive symptoms as measured by the Beck Depression Inventory and cognitive distortion scores across samples of college students, out-patients in therapy for depression and non-depressed psychiatric inpatients.

There is evidence for the relationship between depression and a negative evaluation of experience. For instance, several studies have shown that depressed and nondepressed populations differ on measures of dream content, with depressed subjects reporting more themes of loss and failure (Beck and Hurvick, 1959; Beck and Ward, 1961; Houri, 1976). That depressed individuals show a special readiness to attend to negative aspects of situations is supported by studies of memory in depression (Lishman, 1972; Lloyd and Lishman, 1975). These indicate that one correlate of severe depression is a tendency to recall negatively-toned material more easily and readily than material of a more positive nature. In addition, Lewinsohn et al. (1973) demonstrated that depressed individuals are more sensitive to an aversive stimulus (mild electric shock) as measured by GSR than are psychiatric controls and normals.

Nelson (1977) reported moderate positive correlations between scores on the Beck Depression Inventory and adherence to certain 'irrational' beliefs postulated by Ellis (1962) to account for mal-adaptive behaviours in general. Specifically, the analyses showed that depression was most significantly correlated ($P < .001$) with high self-expectations ($r = 46$), frustration reactivity ($r = 51$), overconcern about the likelihood of future misfortunes ($r = 54$), helplessness ($r = 45$) and the total Irrational Beliefs Score. Another measure of cognitive content, the Automatic Thoughts Questionnaire (ATQ), is described by Hollon and Beck (1979).

Items were derived by asking people to describe specific thoughts associated with depressive experiences and were cross-validated on a separate sample of psychometrically selected depressed college students. The findings indicated that the thirty negative thoughts on the ATQ were subscribed to significantly more often by depressed than by non-depressed individuals. Weissman and Beck (1977 in Rush and Beck, 1979) used the Dysfunctional Attitudes Scale (DAS) to measure specific beliefs relevant to depression. They found a significant correlation between the severity of depression and the degree to which the person endorsed these hypothesised depressogenic schemas.

There is empirical support for the hypothesis that depressed individuals process information in a different manner than non-depressed subjects. Studies have shown that depressed subjects underestimate the percentage of correct feedback they actually receive about their performance on a laboratory task relative to non depressed controls (Demonbreum and Craighead, 1977; Wener and Rehm, 1975). Nelson and Craighead (1977) also found that at a high rate of reinforcement (70% positive, 30% negative feedback) depressed subjects significantly underrated the amount of positive reinforcement as compared to non-depressed subjects. At a high rate of punishment (70% negative feedback), again depressed subjects differed from normals significantly. This time depressed subjects estimated correctly but normals underestimated the amount of punishment. The results suggest that nonresponsiveness to reward in depressed subjects may be a function of distorted cognitive activity. Further evidence for this hypothesis comes from a recent study by Lobitz and Post (1979) who examined the various components

of self-reinforcement in clinically depressed and non-depressed psychiatric patients. While depressed patients demonstrated a lower level of self-reward than non-depressed controls, the results suggested that these low levels of reward may be predicted from the individual's low levels of self-evaluation and self-expectation, i.e. negative cognitions.

Empirical support for the relationship between cognition, mood, and performance comes from the learned helplessness literature. Early work had demonstrated that failure experiences lowered self-esteem in both depressed and non-depressed subjects, though only non-depressed people manifested any positive change in self-esteem as a function of success (Loeb et al., 1964). Moreover, failure experiences were found to have a more adverse effect on a depressed person's expectations of future success than on those of non-depressed people (Loeb et al., 1967). Building on these findings, Miller and Seligman (1973, 1975) and Klein and Seligman (1976) demonstrated that a depressed person's negative expectations about achieving success are not global in nature but are limited to situations in which skill, not chance, is involved. Specifically, relative to a non-depressed group , depressed individuals were found not to change their expectations about subsequent performance on skill tasks on the basis of feedback regarding previous trials.

These investigations have also shown that increases in expectations following successful performance on laboratory tasks correlate highly with subsequent increases in performance levels. Conversely, exposure to uncontrollable failure appears to be associated with reduced performance (Hiroto and Seligman, 1975; Miller and Seligman, 1975; Klein and Seligman, 1976).

In this context, depressed individuals appear to do less well on a variety of psychological tasks than non-depressed subjects (for review, see Miller, 1975). However, it is likely that some of these deficits can be attributed to either cognitive (e.g. negative expectations) or motivational (disinterest in outcome) elements (Hollon and Beck, 1979).

The depressed person's apparent insensitivity to outcome (failure to shift expectancy) has been reported with respect to a carefully diagnosed unipolar inpatient population; this phenomenon was not observed in either the depressed schizophrenic or matched schizophrenic control conditions (Abramson et al., 1978). Considering that one investigation has failed to replicate this result with depressed alcoholics (O'Leary et al., 1978), it may be that the failure to alter expectations following feedback about success is specific to depression and not a characteristic of general psychiatric illness.

According to the reformulated learned helplessness model (Abramson et al., 1978) 'attributional style' may help to explain why depressed individuals appear to process information differently. Several investigations indicate that depressed subjects are prone to attribute negative outcomes, such as failure to internal factors, e.g. personal incompetence (Klein et al., 1976; Kuiper, 1978; Risley, 1978).

These studies all made important contributions in translating Beck's theory into operational terms and then demonstrating a relation between cognition, mood, and performance. However, some aspects of Beck's model have been questioned. For instance, Beck (1976) says that depressed subjects distort their perception

and recall of ambiguous or neutrally-toned environmental feedback. Craighead and Demonbreum (1979) failed to obtain differential perception and recall of neutral feedback among psychometrically identified depressed-anxious, non-depressed-anxious, and non-depressed-non-anxious female college students. Even so, it is doubtful that the selection instruments (BDI and a General Trait Anxiousness Test) were sensitive enough to disciminate accurately such mildly disturbed and heterogeneous groups. As the authors point out, had depressed subjects without anxiety been included in the trial, it is possible that they would have distorted and produced the predicted results which were found in previous studies (Nelson and Craighead, 1977; Demonbreum and Craighead, 1977).

Hammen (1978) has questioned whether all depressives show regular cognitive distortions. Her data suggested the possibility that some depressives have a tendency to distort despite low levels of life stress. In their self-references, such persons may be using the stable self-schema or a negative self concept as Beck proposed. On the other hand, depressives who perceive high life stress show a lesser tendency for cognitive distortion.

Those depressives who are depressed independently of life-stress events may be using negative schema to distort environmental information. Consequently, they may invoke a negative self-schema as a means of giving a systematic negative bias to information about themselves. Negative self-references result. Other depressives, perhaps those who have only recently undergone life-stress, may not use such schema.

Drawing on clinical evidence that depressed individuals describe themselves inconsistently, Davis (1979) has questioned whether a

negative self-schema is a regular symptom of depression. He compared depressed general hospital outpatients with nondepressed normals on semantic and self-reference encoding tasks. As expected, the groups differed significantly on self-reference recall with depressives showing significantly weaker recall on this exercise. However, duration of depressive episode significantly predicted the strength of self-reference recall. Davis concluded that a negative self-schema does not develop until a person has been depressed over a period of time. In a later study which involved tests of free recall with subclinical depressed subjects and normal controls, Davis (1979) provided tentative support for the idea that short-term depressives lack stable cognitive schemas for interpreting personal information. As he acknowledges, the study is not without its flaws (e.g. the use of analogue depressives and 80% of the variance unaccounted for) but it raises interesting questions about the specificity of schemas in relation to different types of depression, an issue not addressed by the cognitive model.

Overall, the results of these studies are compatible with Beck's cognitive theory of depression, though some findings are clearly relevant to other mediational approaches as well (e.g. Seligman, 1976; Rehm, 1977). They indicate that negative thought content and the manner in which information is processed covary with depressed mood and performance but say nothing about the specific causal relationships between these variables; depressed mood could cause depressive cognitions or depressive cognitions could cause depressed mood. However, experiments which show that depressed mood can be induced by manipulating a person's thoughts or attention constitute a stronger basis for Beck's model than correlational

studies.

2. Experimental studies

A number of investigations are available where changes in mood have been induced by changes in thoughts. Velten (1968) had three groups of subjects read aloud and focus on sixty self-referrent statements designed to be depressing, elating, or neutral and two other groups received treatments to control for experimental demand intended to produce simulated elation and simulated depression. Two measures of pre-treatment mood level were obtained from each subject at the beginning of his individual treatment. After treatment seven behavioural measures were taken as criteria for elation and depression. Four of his seven dependent measures discriminated significantly among the treatment groups (writing speed, decision time word association, and the Multiple Affect Adjective Checklist (MAACL). The comparative performances of subjects in the three control groups showed that the obtained mood changes could not be attributable to artefactual effects. Moreover, post-experimental questionnaire data strongly supported the finding that elation and depression treatments had induced elated and depressed mood.

Similarly, Coleman (1975) assigned female college students to one of five treatment or control groups on the basis of scores typical of elation or depression (MAACL), the Weissman-Ricks elation-depression scale (1966) and also on the basis of suggestibility (Howard Group Scale of Hypnotic Suggestibility). The subjects were assigned to one of the five experimental conditions in such a way as to ensure homogeneity of suggestibility and elation-depression across treatment cells. The induction

of positive versus negative cognitions produced significant differences in elation-depression on several dependent variables (MAACL, writing speed, measures of expectation of success, word association, and behavioural measures). Characteristically elated and depressed subjects were able to adopt polarised mood states. The author argued that the data were in line with previous findings that self-esteem is a determinant of elation-depression, and more specifically, that negative self-evaluations are determinants of depression.

These two studies demonstrate a major flaw common to many investigations on the psychological causation of depression (Blaney, 1977), namely that they were conducted on student populations. As mentioned previously, it is doubtful that the mood swings of 'college sophomores' are at all equivalent to those seen in the psychiatric clinic.

Crowley (1976) tried to replicate Velten's findings in a group of depressed inpatients (N = 10) and in a group of university students (N = 20). An attempt was made to control for suggestibility. Using only elation-inducing statements she found significant changes in the depressed group on several dependent measures (2 visual analogue scales to assess mood and anxiety on the MAACL). This effect, however, was not obtained in the normal group. Suggestibility appeared not to account for the effect as the correlations between suggestibility and other subject and control variables were nonsignificant.

Teasdale and Bancroft (1977) used a single subject design to investigate the specific alterations which occurred during 30 second periods of thinking either pleasant or unpleasant

thoughts. They found that this had a systematic effect in modifying the patient's rating of perceived mood and that corrugator EMG was significantly increased by 30 second periods of unpleasant compared to pleasant thinking. It appeared that thought processes on their own can produce changes in at least one physiological concomitant of depression. However, Bonham (1979) has pointed out that the manner in which the negative thoughts were elicited was not controlled, therefore changes in mood may not have been directly related to the thoughts. It is equally possible that mood changes were used as cues to elicit pleasant or unpleasant thoughts.

Thought stopping has been used to reduce the corrugator activity induced by unpleasant thoughts (Teasdale and Rezin, 1978). This appeared to be ineffective as the investigators found that the cognitive changes induced by thought stopping were not matched by the physiological changes. It was suggested that more powerful negative thoughts over longer time intervals might have produced the predicted result.

Blackburn and Bonham (1980) attempted to gain tighter control over the quality and intensity of cognitive content by using the personal thoughts (pleasant and unpleasant) of five depressed patients in a single case design experiment. The main aim was to measure the effect of training in a cognitive strategy ('distancing') which is considered to be a central element in Beck's form of therapy. The pre-test session involved eliciting thoughts related to the patient's experience, rating them on a scale for degree of pleasantness or unpleasantness, and training in the distancing technique, i.e. to regard the thoughts as psychological events which could be subjected to validation, and not as reality itself. For example, if the patient had thoughts that he was a failure, he was to try to 'take perspective', and question the validity of these ideas.

Following this period, cue instructions were given either to 'think the pleasant thoughts', 'distance yourself from unpleasant thoughts', or to 'involve yourself in unpleasant thoughts', i.e. create a vivid mental picture and/or think about them as they normally did.

Mood ratings (by visual analogue) for the three thought conditions were significantly different in the five subjects. The pleasant thoughts evoked the least depression while the 'distanced' unpleasant thought sessions always evoked lower overall mood ratings than the 'involved' unpleasant thought sessions. Interestingly the distancing sessions evoked depressed mood ratings that were lower than the pre-training baseline rating for that thought. The thought conditions were also positively and significantly correlated with corrugator EMG and heart rate across subjects, with distancing being differentiated to some extent by these physiological measures.

The results provide further evidence that thinking negative thoughts can lead to negative affect and, more specifically, that the intensity of mood associated with a thought seems to depend on the way the person copes with the thought and not exclusively on the content of the thought itself. Numerous studies support the hypothesised relationship between dysphoric mood and negative thoughts (e.g. Strictland et al., 1975; Hale and Strickland, 1976; Natale, 1977; Moore et al., 1973; Masters et al., 1979; Rogers and Craighead, 1977). These, in turn, are backed up by a large literature which demonstrates that cognition plays a key role in the labelling and control of general arousal (e.g. Schacter and Singer, 1962; Schacter, 1969; Valins and Ray, 1967; McGuigan and Schornover, 1973; Rimm and Litvak, 1969; Bandura, 1969).

To recapitulate, there is a growing body of data which indicates that affective states can be influenced by cognitive manipulations by

inducing the subject to attend to unpleasant thoughts (see Blaney, 1977). While more research is required to confirm the finding of Blackburn and Bonham (1980), it appears that training in a cognitive strategy that is specific to Beck's therapy can help the individual to modify his affective state once it has been induced.

3. Treatment Studies

The final class of evidence for the cognitive model of depression derives from clinical studies which have compared Beck's Cognitive Therapy to other forms of treatment. Since a description of the procedure appears in the next chapter, only a brief outline of cognitive therapy is presented here.

The ultimate goal of cognitive therapy is the development of rational, adaptive thought patterns. Cognitive therapy progresses through the following stages:

1. depressed individuals become aware of their thoughts
2. they learn to identify inaccurate or distorted thoughts
3. these imprecise cognitions are replaced by accurate, more objective thoughts
4. an essential aspect of cognitive therapy involves consistent therapist feedback and reinforcement throughout the treatment process.

The specific procedures used to attain these therapeutic objectives are both behavioural and cognitive in nature (Wilson, 1978; Beck et al., 1979). For this reason Beck's approach has also been referred to as a 'cognitive-behavioural intervention'.

Although there have been several case studies reported (e.g. Rush et al., 1975) only the controlled studies will be considered in this review.

Several investigations have looked at the relative efficacy of behavioural and cognitive procedures alone and in combination (Gloe, 1975; Shaw, 1977; Taylor and Marshall, 1977).

Shaw (1977) compared group treatments in a population of students who had approached their university counselling centre for help with depression (N = 32). They had to score a minimum of 18 on the Beck Depression Inventory (BDI) and to have experienced depression for at least three weeks. They were then randomly allocated to one of four groups: cognitive therapy; 'behaviour modification', using Lewinsohn's (1970) social skills approach; an attention/ assessment, 'non-directive' control condition and a waiting list group. Treatments consisted of two two-hour sessions per week for four weeks. Assessments were conducted using the Beck Depression Inventory (BDI) and the Hamilton Rating Scale for Depression (HRS-D) which was completed by blind assessors working from a specially edited video-taped interview between the therapist and patient. The results indicated that the cognitive group was most effective in reducing depression as measured by self-report (BDI, $P = < 0.001$) and clinical rating (HRS-D, $P = < 0.02$). At one month follow-up the cognitive and social skills groups were compared again but the continuing trend was no longer significant.

Another group therapy trial has been reported by Gloe (1975, in Weissman, 1979; Rush and Beck, 1979; Beck et al., 1979). Gloe used 10 depressed student volunteers in each of four conditions: cognitive therapy plus 'positive group experience', cognitive therapy alone, positive group experience alone, and a waiting list control group. The combination treatment package is said to have been superior in alleviating depressive symptoms as measured by the BDI.

Taylor and Marshall (1977) attempted to evaluate the various components of treatment by comparing a cognitive, a behavioural, and a mixed approach which they called 'cognitive-behavioural' treatment. Subjects (N = 28) were recruited by advertisement on the university campus. A minimum score of 13 on the BDI and self-reported depression of not less than two weeks was required for admission. Treatment consisted of six individual 40-minute sessions over a four week period. A waiting list control group was also included. The results indicated that the three treatment groups improved significantly more than waiting list controls on all measures (BDI, a visual analogue scale, self-acceptance and self-esteem assessed by repertory grids and the Dempsey D-30 Scale). There were no differences between the cognitive therapy alone and the behavioural intervention alone on any measure. However, the combined group was more effective than either component alone. Thus, the cognitive and behavioural components appeared to be additive in their effects in this small sample of mildly depressed subjects.

These three studies (Shaw, 1977; Gloe, 1975; Taylor and Marshall, 1977) used depressed student populations and must therefore be viewed as preliminary because of important research issues such as the sample size and the severity of symptomatology. Nevertheless, they have been useful in that they employ tightly controlled research designs and provide tentative support for the effectiveness of a specific form of treatment, as do other investigations aimed towards evaluating behavioural interventions (e.g. Fuchs and Rehm, 1977).

While the generalisability of these findings to clinically depressed populations is limited, there is some justification for the growing

confidence in the applicability of cognitive therapy to depressed psychiatric outpatients.

Rush et al. (1977) have conducted the most persuasive investigation to date with respect to the effectiveness of cognitive therapy. Forty-one male and female depressed out-patients, all nonpsychotic (Feighner's Diagnostic Criteria), were randomly allocated to either pharmacotherapy (imipramine) or cognitive therapy. As a group the patients had been intermittently or chronically depressed with a mean period of 8.8 years since the onset of their first depressive episode and 75% were suicidal. Dosage levels for imipramine ranged from 150 to 250 mg per day. Treatment lasted for an average of 12 weeks with a maximum of 20 one-hour individual cognitive therapy sessions and 12 weekly drug therapy visits. The Beck Depression Inventory (BDI) was completed by all the patients. In addition, all patients were rated on the Hamilton scale for depression (HRS-D) and on the Hamilton scale for anxiety. Ratings were made independently of therapists by experienced clinicians but they were not blind to the treatment modality.

Both treatment groups showed statistically significant decreases in depressive symptomatology ($P = < 0.001$). However, cognitive therapy resulted in significantly greater improvement than did pharmacotherapy ($P = < 0.01$) on both the self-rating scale (BDI) and clinical ratings (HRS-D, HRS-A Raskin Scale). Drop out rates were also significantly higher in the pharmacotherapy group. Fifteen out of nineteen cognitive therapy completers demonstrated a clinical remission; five out of the 14 drug completers showed a similar reduction in symptomatology. The response rate to both pharmacotherapy and cognitive therapy surpassed the reported ranges for placebo response

in depressed outpatients (Morris and Beck, 1974).

Attrition in the pharmacotherapy group was significantly greater than with cognitive therapy ($P = < 0.05$): 36 per cent of the patients allocated to the drug group dropped out as compared with 5.7 per cent of the cognitive group. None of the pharmacotherapy drop outs demonstrated any significant clinical improvement and six out of eight eventually reentered therapy. Both groups maintained the treatment improvements at six months follow-up but patients assigned to cognitive therapy showed significantly less depressive symptomatology at three and six months of follow up. When only completers were considered, the cognitive therapy group were still significantly less depressed at three months ($P = < 0.05$) though this trend was no longer highly significant ($P = < 0.10$) at six months follow-up.

This is an impressive study for several reasons. First the authors used operationalised diagnostic criteria to select patients for the trial. Second, Weissman (1979) has pointed out that the patients receiving drugs were not drug failures since their level of response was what might be expected in a drug trial. The fact that cognitive therapy was compared with an active treatment known to generate a high response rate makes the outcome even more remarkable (Hollon and Beck, 1978). Third, the results suggest that for the first time one psychotherapeutic approach may be a more effective short-term intervention in unipolar depression than tricyclic pharmacotherapy.

The most obvious methodological problem is that the two treatment groups differed in the amount of therapist contact. However, the absence of a psychotherapy effect in the Covi et al. (1974) and Friedman (1975) studies (see chapter 2) suggests that this may not

be a critical factor. Becker and Schucket (1978) have argued that the drug levels may not have been optimal for individual patients but they were clearly in line with current practice (Rush et al., 1978). With regard to follow up, chemotherapy was stopped after three months and this might account for the greater numbers of that group having to reenter treatment (Whitehead, 1979). Although the use of blind clinical judges would have been much better, both the clinicians' ratings and self-report measures of depressive symptoms supported the superiority of cognitive therapy. Moreover, it is doubtful that blindness can reliably be maintained in treatment studies comparing drugs with a psychotherapy technique.

Other studies have looked at cognitive therapies in the treatment of psychiatric patients. Rush and Watkins (in press, 1979) assessed outcome in group versus individual cognitive therapy, and the effects of antidepressant medication on attendance and outcome. Forty-four patients were diagnosed as clinically depressed (Feighner's Criteria) and satisfied a description of neurotic depression (DSMII), though a significant number had vegetative symptoms. They had to score a minimum of 20 on the Beck Depression Inventory and at least 14 on the Hamilton scale. Subjects were assigned to one of three conditions: group cognitive therapy, individual cognitive therapy alone, or individual cognitive therapy with antidepressant medication. The protocol specified a maximum of 20 group or individual cognitive therapy sessions over a 10-12 week period, with individual sessions lasting 50 minutes and group sessions 75-90 minutes in duration. Each group contained 4-6 patients and two therapists. Of 44 subjects 28 were given group cognitive therapy, 9 individual cognitive therapy, and 7 were treated with the combination package. Rather than prescribing

a specific drug for the group, an agent most appropriate for each patient was administered.

Post treatment analysis showed that all three groups improved significantly on self-report and clinical ratings. No significant difference was found between individual cognitive therapy alone and individual therapy in combination with medication. However, individual treatment with or without medication was significantly more effective than group cognitive therapy as measured by BDI scores. This finding was not upheld by the clinical ratings and at two months follow-up the differences between groups on the self-report measures were no longer significant. The group cognitive treatment cell had more drop-outs (5) than individual cognitive therapy (1); there were no drop-outs in the combination group.

The authors compared their results with a clinical report on a cognitive therapy group (Shaw and Hollon, 1978) in which only 5 out of 10 depressed outpatients obtained complete remission by the end of treatment. These data are very preliminary but suggest that group cognitive therapy is less effective than cognitive therapy which is given on an individual basis. This is a bit surprising in that the group therapy in Rush and Watkin's study had a higher number of average sessions per subject than either individual cognitive therapy alone or in combination with drugs. Replication of this study using larger samples is necessary before any firm conclusions can be drawn.

Beck et al. (1979) have looked at individual cognitive therapy compared with combined individual cognitive therapy and amitriptyline. The admission requirements for this small study were similar to those in the Rush et al. (1977) trial except that the sample included

schizoaffective patients. Patients were assigned randomly to the treatment groups. Seven out of 33 patients were discontinued from the study, leaving 14 patients in the cognitive therapy alone and 12 in the combined therapy group. Both treatment groups demonstrated highly significant and substantial clinical improvement on self-report (BDI) and clinical ratings (HRS-D) but no between-group differences were found at the end of treatment. Treatment gains were partially sustained at six months follow-up.

As in the previous study (Rush and Watkins, in press), the results appear to indicate that the use of tricyclic medication does not enhance the effectiveness of cognitive therapy administered on an individual basis. However, both studies involved very small samples which, in itself, might account for the lack of differences between the cognitive therapy alone and combined groups. The inclusion of schizoaffective patients in the Beck et al. study also complicates matters. Although patients were allocated randomly to the different treatment conditions, there is the possibility that the two groups differed in terms of diagnosis - a higher proportion of schizoaffective patients in the combined group might account for the relatively inhibited response rate. This is not made clear in their report. What is made clear, however, is that when the schizoaffective patients were excluded from the comparison, the cognitive therapy alone group in this study responded 'approximately as well' as those patients who received cognitive therapy in the Rush et al. (1977) study. On the other hand, it appears that no comparison was made between the combined cognitive therapy and amitriptyline group (minus the schizoaffective patients) and the cognitive therapy group in the Rush et al. trial. Had this been done, it is possible that the combination

package would have done better.

Despite these difficulties, Beck et al. have provided further evidencd in support of the applicability of cognitive procedures to clinical populations.

Other studies have demonstrated the efficacy of cognitive approaches in the treatment of depressed patients (e.g. Morris, 1975; Schmickley, 1976; Maclean and Hakstian, 1978; Dunn, 1979).

Dunn (1979) used a cognitive approach to modify the maladaptive thought patterns of 'depression-prone' psychiatric outpatients. The selection criteria included: a history of treatment of depression, current self-reported depression though not suicidal or psychotic, and they must have been stabilised on low to moderate doses of tri-cyclic drugs (under 125 mg/day). A sample of 24 patients were randomly assigned to either cognitive modification or a medication-support group (of these 20 completed therapy). Patients were seen individually by one of two trained therapists twice weekly for eight weeks. The dependent measures were the Beck Depression Inventory, a frequency count on adaptive nondepressive verbalisations, and a global rating of severity of depression (independent and blind).

Post-treatment and 6 month follow up data indicated that the patients receiving cognitive modification therapy (1) had significantly lower BDI scores ($P = < 0.01$), (2) were rated by judges as less depressed ($P = < 0.05$) and (3) verbalised more adaptive, nondepressive statements ($P = < 0.01$).

A number of criticisms can be made of this study. The population under scrutiny is undoubtedly a very heterogenous group as no effort was made to select patients according to operationalised diagnostic criteria - many types of psychiatric patients are 'prone' to report

feelings of depression, especially anxious patients. The control sample was barely adequate since the patients received the same treatment in this condition as they did when they were being stabilised on tricyclic medication, i.e. drug therapy plus supportive psychotherapy. Moreover, while the length of treatment was controlled, the amount of therapist contact was not. Another worrying aspect of this study is that the drug-support group did not show any improvement during treatment and at follow-up. Assuming that the drug regimen was managed in line with current practice, these patients would be expected to improve significantly in an eight week period. It may be that this group differed from the cognitive modification group in terms of important clinical variables such as duration of illness and response to previous treatments.

Some favourable remarks can be made about this study. First, cognitive therapy is said to be particularly suited to those individuals who suffer from recurrent depressions (Beck et al., 1978). To the extent that these patients were chronic depressives (no descriptive data was reported), the results suggest that the combination of cognitive modification and tricyclic drugs is more effective than supportive psychotherapy plus tricyclic drugs. Furthermore, in terms of the format postulated by Klerman (1975), it appears that individual cognitive modification may exert a positive effect upon pharmacotherapy, whereas supportive psychotherapy appears not to exert this effect. Second, considering the size of the sample (10 in each group) the significance values in Dunn's study are more likely to reflect large, rather than small effects for the treatment. Third, in contrast to many studies which have relied entirely on self-report ratings, Dunn obtained differences between the groups using a behavioural

measure, i.e. frequency of non-depressed verbalisations obtained during a videotaped mental status interview.

In this context, Whitehead (1979) has noted that self ratings may be especially sensitive to change with psychotherapies. Cognitive approaches might be effective in training patients in new cognitive styles (reproduced in self-ratings) but may not necessarily produce a general amelioration of depression. While Dunn's behavioural measure is not immune to experimental demand, the results show that cognitive modification has a measurable effect not only on self-rated depression but on how patients describe themselves, particularly their affective state (more positive self evaluations, more accurate reporting of affect).

Of the studies reviewed, the last four (Rush et al., 1977; Rush and Watkins, in press; Beck et al., 1979; Dunn, 1979) provide stronger support for the use of cognitive (or cognitive-behavioural) procedures in the treatment of depression. All four investigations were conducted using psychiatric patients and three studies (Rush et al., 1977; Rush and Watkins; and Beck et al., 1979) employed operationalised diagnostic criteria in the selection of depressed subjects. However, increased confidence in the generalisability of the results to psychiatric populations is obtained at the expense of experimental rigour. In contrast to the studies which involved mildly depressed subjects (Shaw, 1977; Gloe, 1975; Taylor and Marshall, 1977), these investigations did not control for such factors as attention-placebo, the different components in the 'cognitive-behavioural package', and spontaneous remission.

Taken together, these treatment studies comprise the third source of evidence for a cognitive theory of depression. While

the review presented here is by no means exhaustive (see Weissman, 1978, 1979; Whitehead, 1979; Rush and Beck, 1979; Hollon and Beck, 1979; Hollon and Beck, 1978), it does indicate that cognitive therapy has a place in the treatment of some kinds of clinical depression. Having said that, there is still a long way to go in terms of longer follow-ups, predictive patient and therapy factors, and more importantly, replication of the findings by researchers who are less biased towards the therapeutic techniques under investigation. In the few existing studies of direct comparisons between different psychological approaches (e.g. Shaw, 1977), there appears to be a tendency for the treatment that is the preferred modality of the research group to be shown as more effective by that group (Weissman, 1979).

E. COMPARISON OF COGNITIVE THEORY AND THERAPY WITH OTHER PSYCHOLOGICAL APPROACHES TO DEPRESSION

Beck's model of depression differs from the environmentally-oriented behavioural formulations in two important respects. First, the cognitive model focuses on covert behaviours such as beliefs, attitudes and self-statements rather than overt behaviours. Second, maladaptive thoughts are considered the cause of depression: low mood, reduced motivation, and other depressive symptoms are regarded as secondary manifestations resulting from maladaptive thoughts. In the sense that cognitive theory views depression essentially as a disorder stemming from dysfunctional mediational processes, it is very similar to the Self-Control and Learned Helplessness theories of depression.

The self-control theory addresses many of the same processes discussed in cognitive theory (e.g. selective attention to negative aspects of performance). In comparing the self-control and cognitive models, Rehm

argues that 'the self control model deals with the same phenomena in a way that specifies the distortion processes in operational terms and places them in a theoretical context with other factors in depression . . . the self-control model postulates specific relationships among covert processes and the overt symptomatology seen in depression' (Rehm, 1977, P.800). While there is a distinct emphasis on the role of self-dispensed reinforcement in Rehm's model that is not apparent in Beck's theory, it seems that the self-control theory adds little to existing cognitive formulations. Rehm's theory appears to be a translation of cognitive theory into behavioural terminology (Hollon and Beck, 1979). This in itself constitutes an important contribution to the area and may stimulate research into the cognitive and behavioural determinants of depression.

Beck, like earlier theorists (Freud, 1917; Bibring, 1953), regards low self-esteem as a hallmark feature of depression and offers the concepts of schemas and the negative triad to explain it. A major deficiency in the original learned helplessness model of depression was that it did not explain the depressive's low opinion of himself. However, the reformulated model (Abramson et al., 1978) postulates that depressed individuals who believe their helplessness to be personal (they, alone, are helpless) show lower self-esteem than individuals who believe their helplessness to be universal, i.e. that there is nothing that they or anyone else can do about their helplessness.

In the same context, Beck's concept of schemas has been used to explain why depressed persons are especially inclined to blame themselves for unpleasant outcomes even when their responsibility for the outcome is not at all clear. While the old learned helplessness model did not address this issue, Abramson et al. deal with self-blame in their revised attributional analysis of learned helplessness. In brief, depressives

often make internal, global, and stable attributions for failure and may make external, specific and less stable attributions for their success.

Thus, in attempting to explain low self-esteem and self-blame, the revised learned helplessness model finds common ground with Beck's cognitive model in terms of its comprehensiveness as an aetiological theory. Despite these improvements, the revised learned helplessness theory does not offer any construct to explain why certain people are prone to making depressogenic attributions. In contrast, Beck uses the notion of superordinate schemas (beliefs) to explain why some people are prone to depression while others are not. A big stumbling block in Beck's proposals, however, is that there is very little evidence regarding what specific beliefs are important in depression (Blaney, 1977).

The main caveat in Beck's theory must be that it does not specify what role what cognitions have in what stage of the development or maintenance of what kinds of depressive conditions (Blaney, 1977). Although Beck has not presented a model of the required specificity, his work comprises enough material that could be expected to contribute to such a theory. There already are some promising studies under way aimed at addressing these critical issues (Rush, personal communication).

With regard to treatment, Burns and Beck (1978) have discussed the general characteristics of cognitive-behavioural therapy which set it apart from other kinds of psychological interventions.

Cognitive therapy differs from traditional kinds of psychotherapy both in the manner in which the therapist and patient interact and in the types of problems that are discussed in the interview. In contrast with the'nondirective therapies' (e.g. psychoanalysis or client-centered therapy) the cognitive therapist helps to structure the therapy session and participates actively in discussions with the patient. Beck emphasises

the therapeutic benefits of helping the depressed patient to structure his thinking and behaviour in a systematic, predictable manner.

Another characteristic of cognitive therapy is that the content of the sessions is focused on the here-and-now and less attention is given to developmental and early childhood experiences. As Burns and Beck note,

> 'this is not to say that we feel that the past is not important or that discussions of how past experiences may have contributed to distorted thinking are excluded. However, the major thrust is clarifying the patient's thinking and feelings during the therapy sessions and between therapy sessions' (P.115).

In contrast to the Rational-Emotive approach where the therapist takes a unidirectional posture (e.g. the patient is <u>told about</u> his 'irrational ideas'), the cognitive therapist and the patient work together as a collaborative team. The therapist instructs the patient in a variety of self-help tasks that are performed daily as homework assignments to be done between therapy sessions. The therapist underscores the importance of regular completion of homework assignments which is regarded as a critical element in the treatment process. These are designed to help the patient to recognise and restructure his maladaptive thoughts and behaviours.

Burns and Beck (1978) compare the cognitive approach to behaviour therapy:

> 'Whereas the focus of classical behaviour therapy is modification of the patient's overt behaviour through reinforcement techniques, the primary goal of cognitive therapy involves a transformation of distorted thinking patterns. Thus, the data of greatest importance to the therapist involve the patient's inner experiences, including

his feelings, thoughts, daydreams, and attitudes. The therapist demonstrates that the patient's behaviour is a consequence of his mental state. Behaviour-modification techniques are then used to demonstrate to the patient the irrationality of his beliefs. When the patient assumes more adaptive behaviour patterns, he usually begins to change his attitudes and feelings about himself' (P.115).

Thus, in cognitive therapy behavioural tasks are viewed as experiments which demonstrate to the patient that his rigid, negative self-concept and perceptions of the environment are erroneous. The patient and therapist collaborate in scheduling these experiments which are carried out between sessions. The patient makes detailed observations (and records) about the outcome of each experiment. As he begins to change his behaviour, he evaluates himself and his experiences more realistically.

A second type of homework assignment which differentiates cognitive therapy from other approaches involves the monitoring of specific, maladaptive cognitions. The patient is taught how to record the 'automatic thoughts' that flow through his mind in response to a number of disturbing events. Gradually, the patient recognises that these cognitions are closely connected with his emotions. According to Burns and Beck (1978), 'he then applies a number of tests to determine whether these attitudes are reasonable, logical, and valid. As he learns to analyze these erroneous cognitions, he is taught to restructure his thoughts in a more realistic, reasonable manner' (P.16).

The general strategy behind cognitive-behaviour therapy is to detect thepatient's incorrect thinking and to teach him to assess the degree of his belief in this thinking. The patient and therapist work together in organising specific tests or 'logical demonstrations' that reveal the

discrepancies between the patient's 'automatic thoughts' and reality itself. Once the fallacies and traps in the patient's thinking are uncovered, more accurate appraisals and interpretations of his experiences can be substituted together with more appropriate behavioural responses. The next chapter contains examples of specific techniques that the cognitive therapist uses to counteract the incorrect thinking of the depressed patient.

F. THE PLACE FOR COGNITIVE-BEHAVIOURAL THERAPY

Even if certain kinds of depression can be treated successfully with psychological interventions, why bother to use cognitive-behaviour therapy at all? Why not give antidepressant drugs to all patients? After all, numerous investigations demonstrate that pharmacotherapy is at least partially effective in 70 to 80 per cent of depressed outpatients and is relatively quick in action (Morris and Beck, 1974). In contrast only a few studies show that behavioural or cognitive methods are effective in moderate to severe depressions. In fact, only one investigation suggests that cognitive therapy works better than tricyclic drugs in depressed outpatients (Rush et al., 1977) and there are no published studies which have compared social skills therapy or classical behavioural approaches with drug therapy in the same population. On top of this, compared to drug therapy, even the most time-limited behavioural method involves considerable time and effort on the part of the therapist and patient. Bearing in mind that tricyclic pharmacotherapy has been found to be more effective than group therapy (Covi et al., 1974), marital therapy (Friedman, 1975) and interpersonal psychotherapy (Klerman et al., 1974) in alleviating acute depression, the most logical position for the cognitive behaviour therapist to take is one of realistic humility and openness to empirical investigation.

According to Rush and Beck (1979), 'this lack of supporting clinical research data suggests that it is wise to refrain from promising results with 'any depression' and from contending that behavioural or cognitive treatments are the answer for all depressed outpatients when introducing these treatment methods into a medical setting. Rather, a close collaboration between the descriptive diagnostician, the biologically oriented psychiatrist, and the behaviourally oriented therapist is essential' (P.299).

On the other hand, a balanced view of the situation indicates that there are reasons for treating some kinds of depressed patients with cognitive or behavioural methods (Rush and Beck, 1978). First, these methods may help to prevent relapse in a way that is not available with pharmacotherapy. The purpose of these strategies is to help the patient learn new ways of evaluating himself and the environment and/or specific behavioural skills. Rush and Beck stress that if maladaptive thinking styles remain unchanged, they might make the person vulnerable to future relapse. They cite evidence that drug-treated, remitted patients show cognitive distortions characteristic of the acutely depressed patient (Hauri, 1976). In their six month follow-up analysis, Rush et al. (1977) found that more patients in the drug therapy group had to re-enter treatment compared to the cognitive therapy group ($P = < 0.05$). Certain types of psychological therapies may be better than others in preventing relapse but there is no evidence about this as yet.

Another reason for using cognitive or behavioural methods is that they may reduce the drop out rate from outpatient treatment. On the basis of drop-out data from the Rush et al. study, Rush and Beck project a 5 - 7 per cent drop out rate for cognitive therapy compared to the familiar 25 - 35 per cent rate for pharmacotherapy.

A third reason for investigating specific psychological treatment strategies is that many patients who might be drug responsive either refuse to take the medication because of personal objections or develop side effects which lead them to stop taking the drug (Beck et al., 1978). Moreover some depressives do not respond to antidepressant medication. While some non-responders may require more intense forms of treatment (e.g. ECT, hospitalisation), others might respond well to cognitive or behavioural approaches.

Fourth, cognitive or behavioural interventions may enhance the effectiveness of pharmacotherapy through some interactive process (Klerman, 1975). If these procedures were specifically modified to accommodate drug regimens, they might improve compliance and reduce premature termination from biological treatment.

Preliminary empirical support for a positive interaction between cognitive modification and tricyclic medication comes from Dunn's (1979) study of 'depression-prone' patients, though as discussed earlier, this clinical trial is plagued with methodological difficulties. With regard to compliance, Weissman et al. (1979) found that one kind of role-oriented psychotherapy, namely interpersonal psychotherapy (IPT), in conjunction with tricyclic drugs was more acceptable to patients than either IPT or pharmacotherapy alone. In addition, patients in the combination treatment were significantly more likely to complete treatment ($P = <0.01$). It would be interesting to see if a more structured behavioural treatment could improve upon their compliance rates.

Finally, the use of cognitive or behavioural techniques may pay particularly high dividends in terms of training programmes for mental health professionals. As Rush and Beck note 'Often the psychiatrist or medical trainee is familiar with descriptive diagnostic methods, rating

scales for depression, and biological treatments. However, greater familiarity with day to day behaviour and the recurrent cognitive distortions of the depressed patient allows the trainee to develop a new understanding of this class of disorders and to gauge better the impact of his/her behaviour on the patient. For example, he/she learns to elicit the patient's negative expectations about treatment and to answer themrealistically, thereby increasing compliance with biological or psychological treatments. These educational experiences constitute an important rationale with which the behaviour therapist can enter the medical setting' (P.301).

Thus, there are reasons for guarded optimism concerning the use of specific psychotherapies in the treatment of depression. However, cognitive and less cognitive behaviour therapists wishing to treat depressed outpatients would do well to avoid engaging in what Beck refers to as 'nondimensional/global thinking' with respect to their preferred modalities (e.g. 'cognitive-behaviour therapy is the best way to treat all depressed patients'). In light of the limited clinical research data, it is best to adopt a 'multidimensional schema' such as 'Cognitive behaviour therapy works for some people who present with some kinds of depression'. It is the task of future research to confirm the present findings and delineate the specificity of these procedures across different patients and disorders.

G. SUMMARY OF THE PSYCHOLOGICAL TREATMENT OUTCOME STUDIES

Thus far, this review has covered the physical treatments of depression, the major psychosocial theories that have been put forward to explain it, supporting evidence for these, and in particular, the evidence for the effectiveness of the psychological therapies derived from each theory.

This final section examines the data from the perspective of the psychological treatments as a group and reviews their efficacy alone, in comparison with, and in combination with drugs for the treatment of depression. Table $4G_1$ summarises all of the studies contained in the literature review.

There are two studies (7, 8) testing the efficacy of psychological treatment using subjects as their own controls. Both studies support the efficacy of intensive differential reinforcement schemes (token economies) in the regulation of depressive behaviours.

There are ten studies (2, 3, 5,6, 9, 10, 12, 13, 14, 15) which have looked at the efficacy of psychological treatment in comparison with a specific low-contact,nonspecific,or no active treatment control group. Group Therapy, Marital Therapy, Interpersonal Psychotherapy, Behaviour Therapy, and Cognitive Therapy are represented. Six studies employed nonclinical populations (9, 10, 12, 13, 14, 15). Only five out of ten studies had more than ten patients in each cell. With the exception of one study (9), all of the studies support the efficacy of psychological treatments alone as compared with a control group.

There are five studies (2, 3, 5, 6, 16) which have evaluated psychological treatments relative to tricyclic pharmacotherapy. Contingency management and other forms of behaviour therapy have not been compared with drugs. All of the other psychological therapies are represented and all five investigations have acceptable sample sizes which include only depressed patients. One study (16) found a specific psychological therapy (cognitive therapy) to be more effective than a tricyclic drug (imipramine) in reducing symptoms and attrition in the treatment of acutely depressed outpatients. One investigation (6) found a slightly greater effect for drugs and psychotherapy (Interpersonal Psychotherapy)

in acute symptom reduction and three investigations (2, 3, 5) found tricyclics better than psychotherapy (group, marital, interpersonal) in decreasing relapse rate or symptoms, but psychological treatment somewhat better than tricyclics in the improvement of social adjustment. The effect of psychological treatment in study 5 was evident in patients who stayed in therapy for eight months without symptomatic failure.

The findings on the comparisons of psychological treatments with tricyclic pharmacotherapy indicate the superiority of tricyclics in the treatment of acutely depressed patients. The most persuasive outcome study showing an effect of psychological treatment is that of Rush et al. (1977) but there is an urgent need to replicate their findings before any firm conclusion can be made as regards the efficacy of individual cognitive therapy.

There are eight studies (1, 2, 3, 5, 6, 11, 18, 19) from which information on the combined use of psychotherapy plus tricyclic drugs can be drawn. Six of the studies include depressed patients, one study involved depressed and schizoaffective patients (18), and one investigation (19) tested 'depression-prone' patients. The samples in all of these studies are fairly large. Again, contingency management and behavioural treatments are not included in combination studies but all other psychological treatments are represented. Six out of the eight studies show the superiority of combined treatment over a control group or either treatment alone. On investigation (18) showed an equivalent effect for combination therapy relative to a psychotherapy alone condition. One study (11) shows psychological treatment as superior to combination treatment.

Two other combination studies are available. One study (4) examined the efficacy of psychotherapy (couples group) plus lithium and lithium alone in the long-term management of a moderately large sample of bipolar

depressed patients. The combined therapy was more effective than pharmacotherapy alone. One investigation (17) compared individual psychotherapy (cognitive) plus drug of choice with two different psychotherapy conditions (individual and group cognitive). Although all of the subjects were depressed patients, there were less than ten patients in the individual psychotherapy groups. An equivalent effect was found for combined treatment and individual psychotherapy alone but both were superior to group therapy.

Taken together, the results on the comparisons of combination treatments with a control group or either treatment alone suggest that combined treatment may be the most effective way to alleviate depression.

Various reviewers have covered the efficacy of psychological therapies in the treatment of depression (Weissman, 1978, 1979; Whitehead, 1979; Rush and Beck, 1979; Hollon and Beck, 1978, 1979). These and the review presented in this thesis indicate that there are large gaps in the data. The major limitations in the literature are: (1) heterogeneity in the populations under scrutiny and lack of operationalised diagnostic criteria and standardised clinical assessment of signs and symptoms; (2) relative scarcity of information on bipolar, severely depressed, psychotic and/or hospitalised patients; (3) the lack of psychotherapy control groups in treatment trials of depressed patients (attention-by-assessment, low contact, and nonscheduled 'psychotherapy on demand'); (4) lack of control of spontaneous remission rates (e.g. by waiting list control); (5) the lack of specific outcome criteria (e.g. return to work as a stipulated score on a clinical measure) which would suggest 'real' clinical improvement as opposed to statistically significant improvement; (6) in the case of the nonspecific psychotherapies (Marital, Group, Psychoanalytic, Psychotherapy) the absence of procedural manuals

Table $4G_1$ Evidence for the efficacy of psychological treatments of depression

Study	Treatment groups/ conditions	Sample	Time	Outcome	Researchers
1	Psychoanalytic psychotherapy Imipramine plus psychotherapy/placebo plus psychotherapy	'Neurotic and psychotic depressive reactions and depressions in organic brain syndromes N = 195	1-3 mths	Combination of active drug plus psychoanalytically oriented psychotherapy was more effective than placebo plus psychotherapy	Daneman, 1961
2	Group therapy Imipramine/diazepam or placebo with group or low contact supportive psychotherapy	Moderately depressed, non-psychotic (DSM11) N = 149	4 mths	Imipramine produced more symptomatic improvement than diazepam or placebo, weak effect occurred late for group therapy on perceptions of spouse, empathy and reduction of hostility. No follow-up.	Covi et al., 1974
3	Conjoint marital therapy Marital therapy plus amitriptyline/low contact plus amitriptyline/marital and placebo/low contact and placebo	Primarily neurotic depressives (DSM11) N = 196	3 mths	Amitriptyline reduced symptoms and had an early effect, marital therapy improved family performance and perception of marriage and had a late effect. No follow-up.	Friedman, 1975

Study	Treatment groups/ conditions	Sample	Time	Outcome	Researchers
4	Couples group therapy Couples therapy plus lithium/lithium alone	Bipolar patients N = 65	30 mths	Combined treatment improved post-hospital adjustment i.e. prevented rehospitalisation and marital failure	Davenport et al., 1977
5	Interpersonal psychotherapy (IPT) IPT/low contact with either amitriptyline or placebo or no pill	Primarily recovered neurotic depressed patients DSM11) N = 150	8 mths	Amitriptyline reduced relapse rate; IPT improved social functioning in patients who did not relapse. Combined treatment showed partly additive effect.	Klerman et al., 1974
6	IPT plus amitriptyline/ amitriptyline, IPT, non scheduled treatment	Acutely depressed unipolar non-psychotic (SADS-RDC criteria) N = 96	4 mths	Combined treatment showed partly additive effect, IPT plus amitriptyline resulted in less symptomatic failure. All active treatments were better than non-scheduled treatment. No follow-up	Weissman et al., 1979
7	Contingency Management Tokens plus social reinforcement applied in ABAB reversal design	'Anxious-depressed' inpatient N = 1	4 mths	Tokens plus reinforcement resulted in higher frequency of non depressive behaviours, patient discharged, still out of hospital at 14 months follow-up .	Reisinger, 1972
8	Tokens plus drug therapy, tokens applied in ABA reversal design	'Neurotic depressives', inpatients N = 3	4 days	Token phase resulted in improvement, patients showed adaptive responses in work performance, grooming and social behaviour. No follow-up	Hersen, 1973

Study	Treatment groups/ conditions	Sample	Time	Outcome	Researchers
9	Lewinsohn's therapy Increase pleasant activities/increase protein intake/ monitor activities/ waiting list	Depressed student volunteers (MMPI, Dempsey D-30) N = 30	2 weeks	Pleasant activities and self monitoring groups maintained same level of activity and did better than control groups. Monitor group rated themselves as less depressed than pleasant activities. No follow-up	Hammen&Glass, 1975
10	Behavioural problem solving (social skills) Problem solving/ interest support	Depressed student volunteers (MMPI-D) N = 50	3 weeks	Behavioural group did better than interest support as measured by MMPI D score No follow-up	Shipley-Fazio, 1973
11	Antidepressive Program Meaningless tasks plus group therapy/meaningful tasks, group therapy, imipramine	Neurotic and psychotic (DSM II) N = 56	3 weeks	Neurotics in meaningless tasks plus group therapy improved more than neurotics in meaningful tasks, group and imipramine; no difference in response in psychotic subgroup. No follow-up	Wadsworth and Barker, 1977
12	Self Control Therapy Self control/ discussion-self disclosure waiting list	Depressed volunteers (MMPI-D) N = 36	6 weeks	Both self-control and non-specific treatment groups improved, self-control did best as measured by self-report. At 6 weeks follow-up the differences were not maintained.	Fuchs and Rehm, 1977

Study	Treatment groups/ conditions	Sample	Time	Outcome	Researchers
13	Cognitive Therapy Cognitive therapy/ behaviour therapy (social skills)/ attention by assessment group/waiting list (group modality)	Depressed students referred from counselling centre (BDI > 18) N = 32	1 mth	Cognitive results in fewer symptoms than all other groups, all groups result in fewer symptoms than waiting list. At 4 weeks follow up the differences were no longer significant	Shaw, 1977
14	Cognitive and positive group experience/ cognitive/positive group experience/ waiting list	Depressed volunteers BDI > 13 N = 40	5 sessions in 1 week	Combination was more effective than other treatments. No follow-up data available	Gioe in Rush and Beck, 1979
15	Cognitive and behavioural/cognitive/ behavioural/waiting list	Depressed volunteers BDI > 13 N = 28	6 sessions in 1 week	Combination resulted in the most improvement in depression, all active treatments were better than waiting list. At 5 weeks follow-up the differences were maintained	Taylor and Marshall, 1977
16	Cognitive therapy/ imipramine	Unipolar depressed (Feighner Criteria) N = 41	3 mths	Cognitive therapy showed more improvement and less attrition than imipramine. Differences were maintained at 3 months, but not at 6 months follow-up.	Rush et al., 1977

Study	Treatment groups/ conditions	Sample	Time	Outcome	Researchers
17	Group cognitive therapy/individual cognitive therapy/ individual plus drug of choice	Unipolar depressed (Feighner criteria) BDI > 20, HRS > 14) (Neurotic depression DSM 11) N = 44	3 mths	All groups improved on self-report and clinical ratings; individual and individual plus drug improved to same level, but both were more effective than group therapy as measured by self-report (BDI) At 2 months follow-up the differences were not maintained	Rush and Watkins (in press)
18	Individual cognitive therapy/individual cognitive therapy plus amitriptyline	Unipolar depressed and schizoaffective Feighner criteria N = 26	3 mths	Both groups improved on self-report and clinical ratings but no between-group differences were found. Improvement was only partially sustained at 6 months follow-up	Beck et al., 1979
19	Tricyclic drugs plus cognitive modification/ tricyclics plus supportive psycho-therapy	'Depression-prone' i.e. history of treatment of de-pression, current self-reported de-pression (BDI) N = 20	8 weeks	Cognitive modification and tricyclics was more effective than tricyclics and support measured by self-report, global clinical ratings and behaviour. At 6 months' follow-up the differences still held	Dunn, 1979

that describe the techniques and their sequence during the course of treatment; (7) the absence of longer follow-ups (only four out of the nineteen studies reviewed had follow-ups of six months or more); and (8) the lack of comparisons among the psychotherapies, e.g. cognitive, behavioural and interpersonal psychotherapies. In addition, the testing of these forms of therapy in homogenous groups of patients with other psychiatric disorders is required. This might help to delineate the specificity of these treatments for depression. For example, cognitive therapy might prove useful in the treatment of anxious patients.

H. CONCLUSION

The correlational studies cited in support of Beck's theory appear to demonstrate that depressed subjects (1) differ from nondepressed individuals in terms of cognitive content, and (2) differ from nondepressed people in terms of cognitive processing in such important areas as recall of information, perception of reinforcement and degree of control, expectation of success and/or reinforcement and attributions for success and failure. Problems in these areas might be expected to interfere with comparatively more normal processes of reinforcement. As Hollon and Beck (1979) point out, it does not seem that depressed individuals are either totally impervious to rewards or absolutely unmotivated to achieve success but it does appear that factors in negative cognitive set interfere with these goal-oriented processes.

Likewise, there is support for the hypothesis that (1) thinking negative cognitions can generate negative mood and (2) that negative thinking may interfere with the performance of skilled behaviour. Moreover, a specific cognitive strategy related to Beck's therapy appears to reduce the impact of negative thinking on mood.

It appears that psychological treatments may be effective in alleviating some kinds of depression; that interpersonal, behavioural and cognitive therapies have a measureable effect; and that the most promising form of psychological treatment is Beck's Cognitive Therapy.

It is too early to make any firm conclusions with respect to the efficacy of combined psychotherapy and drug therapy. However, the bulk of the evidence suggests that a multi-modal approach is the best way to treat acutely depressed outpatients. Thus far, the most effective form of psychological treatment (cognitive therapy) and the most effective form of pharmacotherapy (tricyclics) have not been tested adequately in a clinical trial.

The key components in each treatment, the long-term benefits of the treatments (if any), and the specificity of the different therapies across patients and disorders are, as yet, unknown.

CHAPTER FIVE

METHODOLOGY

A. RATIONALE AND GENERAL AIMS

The rationale for cognitive-behavioural treatment of depression is derived from Beck's cognitive model: if the basis of the depression is an overactive set of negative thoughts, then the correction and weakening of these concepts may be expected to alleviate depressive symptoms.

The empirical basis for the application of cognitive theory to the treatment of depression has been detailed in the literature review. Correlational studies show that the preponderance of negative thinking is reflected in the dreams, self-concepts, and attitudes about the future of depressed patients. Studies involving the experimental manipulation of relevant cognitive phenomena indicate a predictable effect on other manifestations of depression such as affect, motivation and performance. Moreover, a cognitive strategy (distancing) based on Beck's cognitive formulation appears to reduce the impact of negative thinking on mood.

Controlled studies of cognitive-behaviour therapy with depressed students and psychiatric outpatients show that it can be more effective than behaviour therapy (social skills), attention by assessment and tricyclic pharmacotherapy. However, one problem with this research is that only one study (Rush et al., 1977) employed operationalised diagnostic criteria in the selection of clinically depressed outpatients. Another difficulty is that it was conducted by the proponents of the

therapeutic techniques under scrutiny, as were subsequent studies directed at examining the efficacy of combined cognitive therapy and chemotherapy (Rush and Watkins, in press; Beck et al., 1979). The need for a replicative study comparing cognitive therapy with pharmacotherapy which involves a research team that is relatively independent of the development of cognitive psychotherapy is apparent.

Combined cognitive therapy and drug treatment has been examined just recently in two controlled trials (Rush and Watkins, in press; Beck et al., 1979) but in each instance the samples were small and, in the Beck et al. study, a very mixed group of patients (depressed and schizoaffective) had received treatment. Though these studies used operationalised diagnostic criteria for selecting depressed subjects, the possible additive effects of the combined treatments were difficult to ascertain because neither study used a drug only control group. Clearly, there is a need to examine the efficacy of combined cognitive therapy and pharmacotherapy in comparison to cognitive therapy alone and pharmacotherapy alone. This would provide a better test for mode of interaction of two treatments which have been shown to be effective in depression (Weissman et al., 1979).

B. SPECIFIC AIMS

The primary objective of the present study is to investigate the scope and limitations of cognitive therapy alone and in combination with pharmacotherapy in the treatment of depressed ambulatory patients.

The specific aims of the investigation are:

1. to assess the therapeutic efficacy of cognitive therapy on its own in the treatment of depressed outpatients,

2. to investigate the effectiveness of cognitive therapy in conjunction with pharmacotherapy as a treatment for depression,

3. to compare the relative effectiveness of cognitive therapy, pharmacotherapy, and combined cognitive therapy/ pharmacotherapy in a broad range of depressed patients in terms of the reduction of acute symptomatology, pattern of response, the minimisation of dropping out of treatment and prevention of subsequent relapse,

4. to evaluate which symptoms respond best to the different types of treatment and which are refractory to treatment,

5. to compare the relative efficacy of the three treatment modalities within and across different clinical settings, namely a general practice and a psychiatric outpatient service.

6. to elicit predictive patient and therapy factors

C. DESIGN

To meet the aims outlined above combined cognitive therapy and pharmacotherapy has to be compared with cognitive therapy alone and pharmacotherapy alone in a factorial group design. Many researchers have discussed the methodological advantages of using factorial designs for the evaluation of treatments in controlled trials (e.g. Kerlinger, 1976; Weissman, 1978 ; Bergin & Lambert, 1978). A 2 x 3 factorial design was decided upon to allow for the investigation of (1) treatment effects, (2) location (or population) effects, and (3) possible interaction effects between the different patient populations and the treatments under observation. In addition, it was intended that the study meet the

requirements of an adequately controlled trial as summarised by Hamilton (1979). These include:

1. Selection of patients according to operationally defined diagnostic criteria,
2. There should be a list of clinical and demographic features in the description of the patients
3. Patients should be separated in treatment groups by an appropriate method of randomisation
4. Careful definition and administration of each treatment is essential
5. The treatments should be defined in terms of the amount given and the length of time required
6. Thorough assessment of the patient's clinical state, i.e. severity of illness, before the start of treatment is necessary
7. Patients should be assessed after a specified period or at regular intervals throughout treatment
8. Finally, there should be follow-up evaluations if this is possible.

No non-specific control group was employed in the design. Given the wealth of adequate placebo-controlled double-blind studies involving antidepressants (e.g. Morris and Beck, 1974), it did not appear scientifically necessary nor ethically justifiable to allocate depressed (and frequently suicidal) patients to a treatment modality (e.g. placebo drug or non-specific psychotherapy control) known to be less effective than antidepressant chemotherapy. The explicit strategy was to consider the basic chemotherapy (plus brief supportive contacts with the prescribing doctor) to be the most appropriate 'control' condition for comparative purposes. Similar arguments in favour of a 'best alternative treatment'

rather than a placebo control are presented by O'Leary and Borkovic (1978) and Hollon (1979) (personal communication).

The major problem with such a strategy is that all active therapies may prove to appear 'equal' at the end of the trial. To put it another way, it may be that no significant differences emerge between the three treatments. Such a finding could occur either because of a genuine comparability in the efficacies of the various therapies, or, because of inadequate implementation of the research design (e.g. unreliable measurement, low statistical power, etc.). Of the controlled trials reviewed, two studies involving homogeneous samples of depressed patients have found significant differences between cognitive-behaviour therapy and other control groups using sample sizes comparable to that anticipated in the present investigation (Rush et al., 1977; Rush and Watkins, in press). Furthermore, the self-report and clinician-rated measures of depression were similar to those used in this study, as outlined below.

It was hoped to obtain at least 30 subjects in each treatment group to make up a balanced design large enough to make group comparisons possible.

D. SELECTION OF PATIENTS

1. Source of referral

Depressed patients were referred from two sources: the clinical population at the Royal Edinburgh Hospital outpatient department, and an ordinary general practice clinic located in Leith, a predominantly working class district in Edinburgh. The Leith practice covers a population of approximately 6,633, of whom 5,700 are below age 65 and mostly from social classes IV and V. Only ambulatory depressed

patients were considered for the trial. There were two reasons for this: first, empirical support for the efficacy of cognitive therapy rests solely on studies which involved mild to moderately depressed populations. Consistent with current clinical practice, these patients might well be regarded as neurotically depressed patients, though it must be emphasised that the term 'neurotic depression' lacks diagnostic specificity and does not, therefore, constitute a distinct diagnostic entity (Akiskal et al., 1978; Kendell, 1975; Paykel, 1971). Given the aims of the research, the existing experimental evidence on outpatient samples, as well as the wish to maintain some degree of comparability across the different findings, it was decided to restrict the investigation to the evaluation of outcome in ambulatory patients only. The second, and more pragmatic reason for choosing to work with an outpatient population was that most depressed patients are treated in outpatient clinics (Goldberg et al., 1975; Clare, 1978; Akiskal et al., 1978).

With regard to the selection of a general practice clinic, it was recognised at an early stage of this research that the role of the general practitioner in the assessment and treatment of psychiatric illness cannot be underestimated. The family doctor occupies a strategic position between specialist psychiatric services and the community. Many investigations have been carried out on the size of the general practitioner's case load, and the concensus of such enquiries has been that between one-tenth and one-fifth of the general practice population are mentally ill or emotionally disturbed (Shepherd et al., 1966).

Neuroses make up a considerably larger proportion of psychiatric morbidity in family practice than hospital psychiatric practice (Clare,

1978). In Shepherd et al's (1966) investigation of 46 London general practices, neuroses comprised 63 per cent and character disorder 4 per cent of the psychiatric disorders seen. Corresponding statistics for all out-patients at the Maudsley Hospital were 40 per cent and 37 per cent and for in-patients 29 per cent and 26 per cent respectively. Only a small proportion of neurotically disturbed patients are treated by psychiatrists and even a smaller proportion by in-patient services. On the other hand, psychotic illness constituted 4 per cent of the total psychiatric morbidity in the survey by Shepherd et al. compared with Mark's (1973) observation that psychoses formed 25 per cent of the Maudsley Hospital outpatient cases and 72 per cent of first admissions to mental hospitals in England and Wales in 1957.

While the majority of emotionally disturbed people are treated solely in general practice, many are referred to specialists and general practitioners form the largest single group of referring agents to the psychiatric services (Robertson, 1979). For example, in North East Scotland almost 78 per cent of new outpatients were referred by their general practitioners (Hall and Hunter, 1970), a figure similar to that provided in a study from the Maudsley Hospital (Hare, 1968) which showed that of 8,000 new outpatients 72 per cent were referred by general practitioners. Kaeser and Cooper (1971) have estimated that in Great Britain at least 150,000 new adult cases are referred to psychiatric services each year from general practice. Thus, there is little doubt that one of the most appropriate places to identify and treat ambulatory depressed patients is in the family practitioner's surgery.

For these reasons and not least because depression is reported

to be one of the most common psychiatric disturbances encountered in general practice (Popoff, 1969; Fry, 1954; Watts, 1956) it was decided that a proportion of patients would be screened and treated in a general practice setting. Moreover, it was hoped that by choosing a practice which serves a predominantly working class area of the city, the sample would include some individuals whom Brown and Harris (1978) have described as being particularly 'at risk' to developing a depressive disorder. This was, however, more a reflection of clinical interest than a specific component of the design.

2. Criteria for selection of depressed patients

i. Definition of clinical depression

A psychiatrist or general practitioner referred patients who appeared clinically depressed from their respective clinics. They were screened by two clinical psychologists (the author and a senior colleague) using a standard psychiatric interview (Present State Examination (Wing et al., 1974)) for elicitation of symptoms and signs. On the basis of this interview clinically depressed patients were defined operationally as people who had met a 'definite' primary major depressive syndrome diagnosis according to the research diagnostic criteria of Spitzer et al. (1978). In addition, case notes were checked for history of illness.

The specific criteria, taken from the third edition of Spitzer's Research Diagnostic Criteria Manual, are as follows:

Criteria for inclusion

1. One or more distinct periods with dysphoric mood or pervasive loss of interest of more than two weeks' duration,

2. At least five of the following symptoms must have appeared

as part of the episode: a) poor appetite or weight loss or increased appetite or weight gain; b) sleep difficulty or sleeping too much; c) loss of energy, fatigability, or tiredness; d) psychomotor agitation or retardation (this as opposed to a subjective feeling of restlessness or being slowed down); e) loss of pleasure or interest in usual activities including social contact or sex; f) feelings of self-reproach or excessive or inappropriate guilt; g) complaints of a reduced capacity to think or concentrate such as slowed thinking or indecisiveness; and h) recurrent thoughts of death or suicide, or any suicidal behaviour.

3. The patient was referred for help during the period of disturbance, took medication, or experienced functional impairment in various social spheres.

4. The first appearance of these symptoms was not preceded by schizophrenia, schizo-affective disorders, panic attacks, phobias, obsessive compulsive disorder, somatisation disorder, anti-social personality, alcoholism, drug use disorder, preferential homo-sexuality, a serious illness which caused major changes in living conditions, or a physical illness known to be associated with psychological symptoms (e.g. thyrotoxicosis).

Criteria for exclusion

In addition to the exclusion criteria of the RDC mentioned above (section 4) patients were excluded from the study if, in the judgement of the interviewer, they presented with symptoms which suggested primary anxiety (i.e. a rating of 0 on item 26 of the Present State Examination) or if an organic brain syndrome,

was suspected.

Moreover, in keeping with the existing evidence for the effectiveness of cognitive therapy, it was decided that patients with a history of bipolar affective disorder would be excluded, as well as those individuals who presented with hallucinations, delusions or other clinical signs which indicated the advisability of inpatient hospitalisation (e.g. extreme agitation or retardation). Also excluded were people suspected of being mentally retarded from clinical judgement.

With respect to medication, those individuals whose medical history contraindicated the prescription of antidepressant medication were screened out of the study. However, patients already taking antidepressants at the point of referral were considered for admission if (1) in the opinion of the referring doctor they had failed to respond to the current drug regimen, and (2) they accepted the possibility that they might be given another type of antidepressant as the treatment of choice.

ii. Minimum level of self-reported depression

Another clinical criterion for admission into the study was self-reported level of depression which had to be at least mild according to the British norms of the Beck Depression Inventory (BDI), i.e. a score > 14. Metcalfe and Goldman (1965) found that British scores tend to be lower than their American counterparts, the respective means being $\bar{x}$ 14.3 $\pm$ 8.3 and $\bar{x}$ 18.7 $\pm$ 10.2. The danger of adopting a low criterion score on a self-report measure is that it might broaden the range of scores so much that it proves difficult to obtain statistically significant differences at the end of treatment, even if patients demonstrate dramatic improvement on other criteria.

However, a relatively low criterion score appeared justified in light of the frequently reported clinical observation that many depressed patients 'don't realise that they are ill' and, when confronted with a 'paper and pencil' instrument, fail to recognise that many of the symptoms on the checklist are relevant to their current state. Previous research has suggested that some types of depressives rated as depressed by psychiatric and ward nursing staff may not endorse syndrome depression items on self-report inventories (Donelly and Murphy, 1976). This argument anticipates the rationale which underlies the use of appropriate observer rating scale in clinical trials.

iii Age

The third criterion for inclusion in the study was age limit: a range of 18 to 65 years inclusive was adopted because of the known difficulties in distinguishing adult psychopathology from childhood and adolescent disturbances (e.g. Forrest et al., 1978). and because of the increasing risk of organic brain syndromes after age of 65 (Welford, 1958, 1967; Post, 1962). No attempt was made to control for age apart from the range given in order not to bias the sample which was meant to be reasonably representative of psychiatric and general practice ambulatory depressed patients.

3. Description of patients

A total of 140 referrals were evaluated. Fifty-two (37%) of the patients assessed were rejected from the study, the proportions for the general practice and the hospital being 43 per cent and 31 per cent respectively. The proportion of patients rejected from the study and the reasons for rejection are summarised in Table $5D_1$.

Table $5D_1$ Patients excluded from the study from the two clinics and the reasons for exclusion

Reason for Exclusion	Hospital Patients N = 22	General Practice N = 30	Full Sample N = 52
Low self-reported depression (< 14 on the BDI)	4	14	18
Alcoholic and/or antisocial personality	4	4	8
Obsessional illness, anxiety, or phobic	5	2	7
Psychotic Depression	1	2	3
Other	8	8	16

Low self-reported depression accounted for 47 per cent of those rejected in the general practice group compared to 16 per cent in the hospital group. In Table $5D_1$ the category 'other' includes a mixture of patients who were excluded for the following reasons: suspected organic impairment or subnormality, secondary depression following a physical or psychiatric illness, bereavement reaction, spontaneous recovery, schizophrenia and tricyclics being contraindicated.

Thus 88 patients constituted the sample at the start of the trial, all of whom met all of the inclusion and none of the exclusion criteria. There were 21 males and 67 females between the ages of 18 and 65. Table $5D_2$ presents the demographic characteristics of the patients from the two clinics. On the basis of their educational background,

Table $5D_2$ Demographic characteristics of patients from the two clinics

Variable	Hospital patients N = 49	General Practice patients N = 39	Full sample N = 88
Sex			
Male	13	8	21
Female	36	31	67
Mean age	43.7 ± 11.2	39.7 ± 9.6	41.9 ± 10.7
Range	21 - 63	19 - 57	19 - 63
Patients with different levels of education			
I	15	29	44
II	12	6	18
III	13	3	16
IV	9	1	10
Patients from different social classes			
I-II	20	5	25
III	24	15	39
IV-V	5	19	24
Marital status			
Single	11	4	15
Married	30	17	47
Separated, Divorced, Widowed	8	18	26

the patients were classified into four groups: level of education less than or equal to school leaving age (level I); at least two 'O' levels and/or must have completed secondary school (level II); specialised training instead of or following 'O' levels on completion of secondary schooling, e.g. business training, technical college, secretarial course (level III); attended University, though they need not have obtained a degree (level IV). Social class was determined according to the patient's occupational status as listed in the 'Classification of Occupations' (1970) booklet published by the Office of Population Censuses and Surveys. In the case of married women, the husband's job was used as the criterion. Single, separated, divorced, and widowed females were classified according to their own occupations. If a patient was unemployed at the time of the evaluation then the job which he/she held last was taken as the criterion for social class.

As should be expected, the sample was strongly biased in the proportion of females to males, women making up 75% of the final sample. The hospital outpatients were slightly older on average, better educated, and from a higher social class than the general practice patients. A considerably higher proportion of general practice patients (46%) had lost a spouse relative to patients referred from the hospital clinic (16%).

With regard to clinical features, the hospital patients had experienced more depressive episodes on average and had been depressed for a longer period than those from the general practice setting. Not surprisingly, over three times as many hospital outpatients had been admitted to a psychiatric hospital at some time in their lives. Sixty-four per cent of the full sample had had previous tricyclic drug treatment.

Table $5D_3$ Clinical features of patients from the two clinics

	Hospital patients N = 49	General Practice patients N = 39	Full sample N = 88
Mean PSE total score	35.1 ± 9.1	29.3 ± 5.8	32.5 ± 8.2
Range	18 - 56	19 - 46	18 - 56
Mean number of previous depressive episodes	3.8 ± 5.6	.7 ± 0.9	2.0 ± 3.3
Range	0 - 25	0 - 3	0 - 25
Mean duration of current episode in weeks	91.4 ± 104.4	36.9 ± 37.7	67.3 ± 85.9
Range	4 - 468	2 - 156	2 - 468
Median duration of current episode in weeks	52.2	28	32.5
Patients with previous psychiatric hospitalisation	25	7	32
Patients with previous tricyclic treatment	41	15	56
Patients reporting suicidal ideation at evaluation (PSE item 25, rated 1)	18	15	33
Patients with prior suicide attempts (PSE item 25, rated 2 or 3)	9	2	11

At the time of their evaluation for entry into the trial, 38% of the total population reported suicidal ideation and another 13% had made a suicide attempt in the recent past (see Table $5D_3$).

Before the full evaluation was given each patient had to be at least mildly depressed, defined by a minimum score of 14 on the Beck Depression Inventory (see 'Measures used' below). Table $5D_4$ shows the means, standard deviations, and ranges for each of the patient groups and for the two populations combined.

Table $5D_4$ Beck Depression Inventory Scores at initial evaluation for patients in the two clinics

	Hospital Patients N = 49	General Practice N = 39	Full Sample N = 88
BDI score:			
Mean	26.4	25.7	26
S.D.	7.6	6.9	7.3
Range	15 - 41	14 - 43	14 - 43

The mean score on the BDI for the full sample at intake indicated moderate depression ($\bar{x} = 26$) which is consistent with the British norms for the Beck Depression Inventory; the mean score for the hospital patients was just slightly higher than that in the general practice group.

The aim of admitting at least 30 patients into each treatment group was fulfilled except for the drug treatment group. Only 28 patients were admitted to this group. This was due as much to the randomisation procedure as it was to the purely practical problem of

a time constraint relative to the resources available for the research.

Of the 30 patients admitted to the combined cognitive therapy and pharmacotherapy group, eight patients dropped out of treatment, leaving a total of 22 completers. These figures were identical for the cognitive therapy group. In the pharmacotherapy group, eight out of 28 patients dropped out of therapy, leaving a total of 20 completers. Attrition was higher in the sample of general practice patients.

E. DESCRIPTION OF TREATMENTS

1. Cognitive therapy alone

Cognitive therapy alone consisted of at least one weekly 60 minute session with the treating clinical psychologist. No psychotropic drugs were allowed. The nature of the specific psychotherapy, as described in an unpublished (Beck et al., 1978) and published manual (Beck et al., 1979), was short-term and focused on correcting the patient's negative thinking. The treatment protocol did not specify a maximum number of interviews though the longest time a patient could remain in therapy (and still be included in the trial) was twenty weeks. Clinically, therapy was stopped sooner or later according to individual cases.

Cognitive therapy techniques are designed to identify, reality test and correct maladaptive distorted conceptualisations and the maladaptive schemas (beliefs) underlying these cognitions. The idea behind the approach is that the patient learns to master problems and situations which he previously considered insuperable by re-evaluating and changing his thinking. The ultimate goal is to help the patient to think more realistically and adaptively with a view to reducing symptoms.

Table $5E_1$ shows a summary of the steps used (for a detailed description see Beck et al. (1979)). These consist of a detailed assessment phase and a certain amount of didactic, confrontation, and behavioural methods - all however aimed at a main target, the cognitive element. The purpose of therapy is cognitive restructuring, using Kelly's (1955) notion of the patient and the therapist as scientists who collaborate in investigating the patient's personal constructs.

Step 1 consists of an assessment phase and requires the application of a behavioural, or problem-oriented conceptual framework to depressive disorders. The patient's symptoms may be regarded as a set of target problems which manifest at several levels of functioning - cognitive, behavioural, or physiological. The target problem may be defined as any of the components of the depressive disorder that involves suffering or functional disability. Breaking down the depressive syndrome into component problems facilitates goal directed activity for the patient and therapist alike and is indispensable for structuring therapy within individual sessions and throughout the course of treatment, e.g. establishing agendas for each session and planning homework assignments.

Table $5E_2$ summarises the major components of the depressive syndrome and how these may be subdivided into specific target problems.

According to Beck et al. , the therapist in collaboration with the patient makes a determination as to which of the target problems should be dealt with on the basis of many factors, e.g. which are most distressing to the patient, which are most amenable to therapeutic intervention.

For the sake of brevity it is not possible to discuss all of the specific techniques for dealing with these problems. In general,

Table $5E_1$ Steps in cognitive therapy

1. Assessment phase
   i.e. breaking down the depressive syndrome into component problems

2. Socialisation for therapy
   i.e. providing a rationale for the cognitive approach

3. Monitoring of automatic thoughts

4. Distancing

5. Identification and discussion of stereotyped themes (e.g. loss, failure, hopelessness)

6. Elicitation and challenging of depressogenic beliefs

7. The use of behavioural techniques
   (e.g. graded tasks, diary of waking activities, triple column technique)

Table $5E_2$

Assessment Phase

Break down syndrome into 'problems'

Affective	Motivational	Cognitive	Behavioural	Physiological
1. sadness	1. loss of positive motivation	1. indecisiveness	1. passivity	1. sleep disturbance
2. guilt	2. increased dependency	2. problems seen as overwhelming	2. avoidance	too much
3. shame		3. self criticism	3. inertia	too little
4. anger		4. concentration and memory difficulties	4. reduction in social skills	2. eating
5. anxiety				too much
				too little
				3. loss of libido

Advantages: facilitates goal-directed, structured therapy within and across sessions

the techniques may be classified as

A. predominantly cognitive in which the major focus is on the patient's thinking, and

B. predominantly behavioural which consists of engaging the patient in specific activities or projects which, in themselves, help to relieve some of his suffering and will have a generalisation effect onto the other problems.

Step 2, socialisation for therapy, is didactic and involves the discussion of certain basic principles of epistemology. The attitudes conveyed are that a perception of reality is not reality itself, but only a rough approximation. The patient's sampling of reality is restricted by the inherent limitations of his sensory function, and secondly, his interpretations of his sensory input are dependent on inherently fallible cognitive processes. Physiological processes can substantially alter perception and comprehension of reality. Thus, the patient has to accept the distinction between external reality and psychological phenomena and that reliable knowledge depends ultimately on having access to enough information. The influence of thinking on mood is explained with examples (see Beck et al., 1978, 1979) - in particular examples of 'maladaptive automatic thinking' which precedes a feeling of dysphoria.

Beck emphasises the importance of this stage of treatment, in particular helping the patient to recognise the connection between thinking and feeling. Beck suggests that the therapist may explain the cognitive approach to the sophisticated patient in the following way: 'when depressed persons consider their experiences, they generally pay most attention to the negative meanings which can be attributed to

these events. When this occurs, the negative thinking feels realistic to you. The more believable this negative thinking is the more upset you will feel.' (Beck et al., 1978, P. 98).

Throughout treatment, but especially during the initial phases, the therapist repeatedly focuses on the connection between feeling and thinking. For example, if the patient says 'I feel sad, empty' the cognitive therapist will ask 'what are you thinking about?' Although this step in therapy is mainly didactic, the therapist, by his interviewing technique, employs modelling as an important component in teaching the cognitive approach.

After the cognitive approach has been explained and discussed, the third step, the monitoring of thoughts, or 'filling in the blank' begins. A basic procedure for helping the patient identify his automatic thoughts is to train him to observe the sequence of external events and his reactions to them. The patient may report a number of circumstances in which he felt upset. The emotional upset becomes understandable if he can recollect the thoughts that occurred during that gap. Figure $5E_1$ illustrates the basic idea behind the technique. Both examples have been taken from the casenotes of a patient who received only cognitive therapy in the present study.

Figure $5E_1$

	Event and/or memory of event	Automatic thought	Emotion
Example 1	Pending divorce	'I failed in my marriage'	Sadness
Example 2	Recalls the decision and act to abort first child	'I'm a murderer' 'A killer can never be a good mother'	Guilt

The next step (4) in treatment is called distancing. Some patients who have learned to identify their automatic thoughts recognise their unreliable and maladaptive nature spontaneously. Distancing refers to the process of regarding thoughts objectively, that is, seeing them as psychological phenomena, not as identical with reality. Recent experimental work indicates that this component of Beck's therapy has a measurable effect in reducing the impact of negative thinking on mood and its psychophysiological correlates (Blackburn and Bonham, 1980).

The monitoring of automatic thoughts and distancing from them help to bring out the stereotyped themes that pervade the patient's thinking: for example, themes of inferiority, inadequacy, guilt. In step 5 the therapist points these out and challenges them by indicating the lack of evidence, by offering contrary evidence and by underlining the overgeneralisation or arbitrary nature of the conclusion. The patient is encouraged to put forward alternative hypotheses and draw alternative conclusions (cognitive reappraisal and alternative therapy).

A common theme in the thinking of depressed patients is hopelessness, a cognitive symptom which is reported to correlate highly with suicidal intent (Minkoff et al., 1973).

The particular therapeutic strategy used in dealing with the patient's hopelessness is based on the premise that he/she is locked in by his arbitrary conclusions. It does not occur to him to question these conclusions (automatic thoughts), e.g. 'there is no point in living', 'I have nothing to look forward to,' or 'I am a burden to my family and they will be better off without me'. As far as the patient

is concerned these thoughts and their themes appear very reasonable, even when they are questioned by the therapist. However, according to Beck et al. (1978) the adoption of a problem solving approach (e.g. exploring the validity of the patient's fixed ideas, or by asking the patient to think rationally about the advantages and disadvantages of suicide) facilitates the break up of the 'closed' cognitive network. As the patient reflects on the evidence bearing on his negative thinking, he/she may recall information that contradicts the fixed belief. Beck et al. (1978) give the example of a woman who originally said that she had never been happy but, when confronted with evidence that failed to support this, was able to recall a period in her life, albeit during her adolescence, when she did experience contentment.

The next step (6), eliciting depressogenic schemas, often takes place in conjunction with the discussion of automatic thoughts and their related themes. While talking about the themes, the 'list of tyrannical shoulds' or rules that the patient uses to regulate his life or appraise other people become apparent. These rules are often unrealistic, or are used inappropriately or excessively. Typical unrealistic rules are, for example, 'in order to be happy, I need to be loved by everybody, therefore I should make everybody love me' or 'I should always be strong and never show weakness or get sick'; another example would be 'I should succeed at everything I do'. These schemas (beliefs) can be pinpointed and challenged by the therapist (confrontation). Beck et al. note that once several rules have been identified one technique, which they call a variation of response-prevention, may be used to modify the 'should'. The therapist guides the patient to 1) verbalise the 'should', e.g. 'I should never get

angry with people', 2) predict what would happen if he did not follow this inner command, e.g. 'my colleagues will reject me completely', 3) carry out an experiment to test the prediction, e.g. verbalise a well-practised, but low level assertive response, and 4) according to the results of the experiment revise the 'should', e.g. record his colleague's reaction in a diary which might turn out to be mild annoyance rather than outright rejection.

The emphasis here, as with all of the techniques of cognitive therapy, is to get the patient to adopt an experimental approach to his difficulties and to test the negative thinking (hypothesis) which is causing him so much distress.

Finally, in keeping with the experimental approach is the use of behavioural techniques which complement the intellectual procedures in cognitive therapy. These may include a hierarchy of graded tasks (or success therapy) used to challenge the patient's cognition that he cannot succeed at anything, also to increase his activity level and thus change his self-image and simultaneously change other people's opinion of and attitude to him. Keeping an hourly diary of waking activities (see Appendix I) which he marks for mastery and pleasure can change the patient's poor self image and focus his attention on the positive aspects of his life instead of the negative, thus altering his negative abstractions. The patient also usually keeps a log of automatic thoughts. The specific technique consists of writing down the situation that elicited the unpleasant feeling in one column, writing down the negative cognition in another column and the rational response in another (triple column technique, see appendix II). Beck also uses modelling techniques such as role

reversal: the therapist, for example, plays the role of the patient as he sees himself: inadequate, inert, weak. The patient is coached to assume the role of the therapist: understanding,yet directive and more objective about the patient's difficulties.

Thus, in addition to a didactic approach and socratic dialogues where the therapist puts forward an opposite viewpoint and by argument he and the patient can work towards a realistic assessment of a situation, the patient has a fair amount of homework to do. Graded tasks, keeping a diary, monitoring automatic thoughts and, as seen in the example of modifying the 'should', carrying out specific behavioural assignments to improve interpersonal problems are all likely to be included in the patient's therapy programme.

Tables $5E_3$ from Beck (1976), P. 275-278, list the target approach to depression and recommended techniques to tackle them.

2. Pharmacotherapy alone

Consistent with clinical practice and to maximise the effect of antidepressant medication, the treating physician selected an agent most indicated for each patient at hand (Baldessarini, 1977). Medication consisted of tricyclic or tetracyclic antidepressants (N = 57 completers only), monoamine oxidase inhibitors ((MAOI) N = 2 completers only), lithium (N = 1, completer only) and Anafranil (N = 4, completers only). Tricyclic medication consisted of 100 - 200 mg/day of amitriptyline or imipramine administered in a flexible dose which was raised to 125 mg within ten days, stabilised over the next two weeks and maintained for another 12 weeks. As with the cognitive therapy group, the maximum time allowed was 20 weeks. Tetracyclic medication (Mianserin, Zimelidine)

Table $5E_3$ The target approach to depression (from Beck, 1976, 375 - 378)

Specific problem area (target)	Reasons given by patient	Therapeutic approach
1. Behavioural symptoms a. Inactivity b. Withdrawal c. Avoidance	1. Too tired or weak 2. Pointless to try 3. Will feel worse if active 4. Will fail at anything I try	1. Probes a. What lost by trying? b. Has passivity done any good? c. Will feel worse if passive d. How do you know? 2. Activity schedule 3. Graded task assignment 4. Cognitive rehearsal
2. Suicidal wishes	1. No point to living 2. Too miserable, need escape 3. Burden to others 4. Cannot cope with obligations/problems	1. Expose ambivalence a. Question reasons for dying b. List reasons for living 2. Alternative therapy a. Alternative views of problems b. alternative actions 3. Reduce problem to manageable units
3. Hopelessness	1. Nothing will work out 2. Same as suicidal 'reasons'	1. Empirical demonstration of fallacy of negative predictions 2. Question 'reasons'

Specific problem area (target)	Reasons given by patient	Therapeutic approach
4. Lack of gratification	1. Cannot enjoy anything 2. No satisfactions 3. Activities do not mean anything	1. Remove 'blinders' 2. M and P Therapy: Look for gratifications and label them 3. Explore meaning of goals 4. Counteract 'killjoy thoughts
5. Self-criticisms	1. Am defective, weak, etc. 2. I should be more adequate 3. Am responsible for problems	1. Identify and reason with self-criticisms 2. Role play: self sympathy 3. Discuss: 'tyranny of shoulds' 4. Triple column technique
6. Painful affect	1. I can't stand the pain 2. Nothing can make me feel better	1. Distraction 2. Raise threshold by ignoring affect 3. Counteract with humour, anger 4. Induced imagery 5. Triple column technique
7. Exaggeration of external demands, problems, pressures	1. I am over-whelmed 2. There is so much to do I can never do it	1. Problem resolution a. list things to do b. set priorities c. check off accomplished tasks d. concretise and split up external problems 2. Cognitive rehearsal

consisted of 60 - 90 mg/day and 100-200 mg/day respectively. Monoamine oxidase inhibitor treatment consisted of phenelzine 60 - 90 mg/day; lithium carbonate was used to obtain a serum level of 80 - 120 mmol/l. No other medications were prescribed for these subjects. Patients treated in the general practice clinic received either tricyclic or tetracyclic medication; MAOIs and lithium were not prescribed in this group.

It was decided that a period of 12 weeks was well beyond the time when a maximum effect to drug therapy could be expected. In fact, considerably shorter time periods have been reported in many successful drug trials (e.g. Morris and Beck, 1974).

In addition to taking antidepressant medication each patient received supportive counselling from the attending physicians.

3. Combined cognitive therapy and pharmacotherapy

Cognitive therapy plus drugs consisted of both treatments as described above.

4. Therapists and therapists' training

Two therapists participated in the study: two qualified clinical psychologists, one of whom is a principal psychologist attached to the MRC Brain Metabolism Unit, Thomas Clouston Clinic, Royal Edinburgh Hospital, the other being the author, who had a level of clinical experience equivalent to that required for eligibility as a senior psychologist in the National Health Service. Each had considerable experience in the treatment of adult psychiatric populations.

Beck et al. (1979) have given a summary of the pre-requisites needed to be a cognitive therapist. These are taken directly from the

manual. The cognitive therapist should have:

a) 'a clear understanding of the cognitive model of depression'

b) 'a grasp of the conceptual framework of cognitive therapy (as outlined in various texts) as well as its special application to the treatment of depression'

c) 'formal training at a centre for cognitive therapy. This should include supervision in the therapy of depressed patients'.

d) 'training at workshops, group preceptoring and institutes and the utilisation of videotapes and annotated transcripts. Such training also requires continual supervision by a qualified instructor on a weekly basis until the competency criteria are met'.

(from Beck et al., 1979, 25-26)

The two psychologists involved in the present investigation had familiarised themselves with the cognitive theory of depression as described by Beck (1967, 1973, 1976), the special application of the theory to the treatment of depression, as well as the procedures outlined above. Both the theory and procedures of the cognitive approach to depression were discussed thoroughly, with special emphasis accorded to techniques for the prevention of suicide. Prior to treating patients, the therapists experimented with the various methods, one playing the role of a depressed patient while the other acted out the role of a cognitive therapist.

In addition to using the manual 'Cognitive Therapy of Depression (unpublished version, Beck et al., 1978) as a guide, videotapes of an experienced cognitive therapist (A.T. Beck) were viewed and the demonstrated techniques discussed in detail with the aid of explanatory transcripts. Moreover, throughout the self-training process and prior

to the start of the actual study, each of the therapists had treated at least two depressed inpatients and several outpatients, some of whom had been prescribed antidepressant medication.

Neither of the therapists had received formal training from the developers of the cognitive therapy techniques. This was due to several practical difficulties.

When the project was planned originally, no formal guidelines for therapist training had been developed. Another problem was that, while it was theoretically feasible to receive some degree of training from the developers of cognitive therapy at the University of Pennsylvania, the resources available to the present researchers precluded extensive training for two therapists in an overseas academic centre. A third difficulty was that at the time the project was being planned, there were no facilities available in Great Britain which could have offered suitable training in the form of seminars or workshops.

In an attempt to control somewhat for the quality of therapy being administered, audiotaped recordings of selected therapy sessions were reviewed regularly by the therapists. Some of these were subsequently evaluated by experienced cognitive therapists at the Centre for Cognitive Therapy (see appendix III). The evaluations provided by the Pennsylvania staff indicate a reasonable degree of proficiency on the part of the author in the administration of cognitive therapy.

Having said that, it is recognised that the therapeutic skills of the therapists would have been enhanced by more formalised training.

F. PROCEDURE

Table $5F_1$ shows the design of the study.

1. Patients referred from the two sources, prior to any evaluation, agreed that if eligible for the study they would accept pharmacotherapy, cognitive therapy or a combination of the two. They were advised of the nature of the study and the available treatment modalities and that acceptance into the study precluded the concurrent use of psychotropic medications other than that prescribed by the doctor for purposes of the research. The nature, potential risks, and benefits of the research were discussed. Each patient was free to leave the study at any time.

2. The first criterion for admission into the study was level of depression which had to be at least mild according to the British Norms of the Beck Depression Inventory (BDI $\geq$ 14).

3. Those patients who met the criterion score on the BDI were then screened using a standard psychiatric interview, i.e. the Present State Examination (Wing et al., 1974), for elicitation of symptoms and signs. Two clinical psychologists conducted the interviews, both of whom had received training in the use of the Present State Examination. On the basis of this interview, Spitzer's research diagnostic criteria were checked (Spitzer et al., 1978). The patients had to present with a symptom profile which satisfied the criteria for primary major depressive disorder as outlined above. Those patients in whom anxiety was the primary problem were excluded from the trial.

4. After acceptance into the study, a series of baseline measures

Table $5F_1$ Design of the study

Referral Source	Screening	Assessment	Admission to trial	Treatment	Follow-up
1. Hospital out-patient department 2. General Practice in Leith	1. Accept if BDI score > 14 2. Present State Examination 3. Spitzer's criteria for primary major depression	1. Hamilton Rating Scale for depression (HRS) 2. Hopelessness Scale (GES) 3. Alternate Mood Scale (IDA) 4. Semantic Differential 5. Counting speed 6. Writing speed	Random allocation to Cognitive therapy, Drug of choice or a Combination of the two	1. Administered in the two clinics 2. Drop out if no response at 10-12 weeks (50% on HRS or BDI) 3. Stop at ≤ 20 weeks	Assessment every six weeks for one year using same battery

were taken, i.e. several self-report scales, an observer rating, and two behavioural tasks.

5. The patients were then randomly allocated to one of three treatment modes: cognitive therapy alone, pharmacotherapy/drug of choice, or a combination of the two. Treatment assignment was made by opening a sealed envelope which contained the actual treatment assignment. Separate sets of envelopes were prepared for each location, i.e. a total of 45 envelopes for each clinic, each with an equal distribution of the three treatments. To ensure equal allocation of different levels of depression, three levels of severity were controlled in the random allocation, mild (BDI 14-20), moderate (BDI 20-26), and severe (BDI 26+). All of the envelopes were prepared prior to any patient selection. When a patient became eligible for admission to the study, the interviewer contacted the appropriate secretary at the clinic who then selected an envelope at random, thereby allocating the patient to the treatment mode. This procedure, in particular ensuring that the treatment envelopes were in the possession of a staff member not connected with the study, allowed for proper randomisation of treatment. For the hospital patients, the consultants in charge reserved the right to veto the admission into the study of any single patient if he had been randomly allocated to a treatment mode which was undesirable from the consultant's viewpoint. However, this eventuality never occurred.

6. Treatment was administered in the two clinics with both cognitive therapy groups attending usually twice a week at the beginning and once a week thereafter.

7. Re-assessment on all baseline measures except the Present State Examination was done every two or three weeks if this was possible. Patients who received pharmacotherapy alone had a therapist contact only at the time of re-assessment. They were seen by the prescribing psychiatrist or family doctor and by one of the two clinical psychologists involved in the trial, their role being simply to distribute the self-report scales and to administer the two objective behavioural measures.

8. If no response occurred after a maximum of 12 weeks of treatment patients were dropped from the trial whatever type of treatment they were on. Response was defined, albeit somewhat arbitrarily, as a decrease of 50% or more on either or both of the two main depression rating scales, i.e. the Beck Depression Inventory and the Hamilton Rating Scale. It was felt that a 50% reduction in either observed or self-reported depression would reflect a degree of 'real' clinical improvement from the patient's viewpoint (see Klerman et al., 1974).

9. A maximum of 20 weeks of therapy was specified, though clinically treatment was stopped sooner or later according to individual cases.

10. After treatment the protocol specified that patients be followed up every six weeks for one year using the same measures. This part of the study is still in progress, therefore follow-up data will not be included in the reporting of results, this study being concerned only with the outcome of treatment.

G. MEASURES AND RATING SCALES

Research studies in depression call for means to estimate reliably and validly the severity of illness in the patients being examined.

This need is second only to that for uniform diagnostic criteria and is important for matching groups of subjects within a given unit as well as for comparing the results of biological, pharmacological, and psychological studies carried out in different centres (Carrol et al., 1973).

The measures used in this study were chosen because they are, on the whole, well documented measures of aspects of depression which this study sets out to investigate.

Although the reliability and validity of some of the measures is less than adequate, the instruments are widely used in psychiatric research, thus making results relatively comparable and repeatable for other investigators. The measures and rating scales will be described under the subheadings of the functions they measure.

1. Diagnostic

According to Rush (1976), 'diagnosis (to know through) represents a hypothesis, the clinician's best hypothesis, about the nature of an ailment. It designates both what the ailment is and how it came about. Specifically it focuses on the nature of pathological processes or the nature of the malfunctions presumed to occur. It is a statement of both what is the phenomenon and how one explains the phenomenon. Thus, diagnosis involves an understanding based upon classification and explanation' (P.47).

Although many physicians would agree with Dr. Rush on the nature of diagnosis, it is far more difficult for them to reach a concensus on what exactly constitutes a depressive disorder.

The problem of diagnosing the depressions was referred to briefly in the introductory chapter (chapter 1) in relation to the multiple meanings of the term, both in verbal and written communications about

the phenomenon. It may be an affect, a symptom, a syndrome or an illness (Mendels, 1975). Other factors contributing to difficulties in diagnosis include: the failure to operationalise consistently the criteria for making a diagnosis of depression; the inability to make a distinction as to when depression ends and another psychiatric disorder begins ('boundary problem'); and most importantly, the problem of poor interrater reliability (Rush, 1976; Zubin, 1967; Kendell, 1976).

In an effort to resolve these difficulties, in particular low diagnostic reliability, Wing et al. (1974) developed the Present State Examination, the main instrument used to screen referrals for the present investigation.

(i) Present State Examination

The Present State Examination, or PSE, (Wing et al., 1974) was used for the purpose of eliciting symptoms, those psychological, behavioural, and physical phenomena which have generally been thought by psychiatrists to constitute mental illness. It is a structured psychiatric interview the development of which as an aid to the differential diagnosis of the mentally ill has been well-documented (Wing et al., 1965; Wing, 1970, Wing, 1971; Wing et al., 1974). In its brief version, it has been shown to be convenient and acceptable for use in population surveys (Wing et al., 1976; Wing et al., 1977; Duncan-Jones and Henderson, 1978; Henderson et al., 1980).

The manual of the ninth edition of the Present State Examination (Wing et al., 1974) provides all the necessary information on the rationale behind the development of the PSE, the derivation of items, the administration of the interview, and the use of the scoring key.

Essentially, the interview is a checklist of 140 items which systematically covers all of the phenomena likely to be considered during a psychiatric examination (see Appendix IV). The interview is completed by rating the presence or absence of each symptom. Most of the symptoms are scored 0, 1, 2, 8 or 9. A rating of 0 means that the symptom was absent during the past month, (8) that the interviewer was uncertain even after proper enquiry, whether the symptom has been present during the past month and (9) that no decision can be made because the question was not asked as the patient did not answer or replied incomprehensibly. Ratings of 1 or 2 indicate whether the symptom was present in moderate or severe form.

A major advantage of the ninth edition is that each symptom is described in great detail in a glossary of definitions. Wing et al. emphasise that training with this glossary is essential if the ratings of symptoms are to be made in a uniform or reliable way. Both of the clinical psychologists who conducted the interviews in this study were trained in its administration, not by the developers of the PSE, but by one of their delegates, a consultant psychiatrist at the Royal Edinburgh Hospital.

The present State Examination can be used in a number of ways for scientific research. According to Wing et al. (1974), 'the first and most obvious use of the system is to describe, in terms of symptoms, syndromes or classes, the psychopathological characteristics of groups of people at a defined point in time' (P.32). Individual or group syndrome profiles using their Catego classification system allows for the delineation of differences between classical

diagnostic groupings (e.g. schizophrenia, psychotic depression) and comparability across different selected psychiatric populations and cultures (Cooper et al., 1972; WHO, 1973, in Wing et al., 1974).

The instrument has also been used to measure change in symptoms. For instance, the ninth edition of the PSE was used in two trials of preventive medication in schizophrenia (reported in Wing et al.) while an earlier version (eighth edition) was used monthly to assess change in an investigation of two antidepressant drugs (Lipsedge et al., 1971, in Wing et al., 1974).

Apart from classifying items into different psychiatric syndromes and measuring change over time, the PSE can be used to standardise the description of mental state of patients included in various research projects. Many of the treatment studies reviewed in the previous chapter, including drug trials, suffer from the fact that their results are not comparable with those undertaken at different sites, particularly in terms of the populations studied. The main reason for choosing the PSE in this investigation is that it is a useful aid to the standardisation of selection of depressed patients.

Data are available from two studies in which the PSE was used on a large scale, the US-UK Diagnostic Project and the WHO International Pilot Study of Schizophrenia (IPSS). The inter-observer reliability ratings of individual items has been examined extensively in these projects and, with only a few exceptions, high levels of reliability have been reported (Wing et al., 1974). For example, in the IPSS tests of item reliability were carried out between nine research centres. The analysis included data obtained from simple investigation of inter-observer reliability on both live and videotaped interviews. The

The range of values of the intraclass correlation between centres was from 0.97 to 0.43 with a median of 0.77. Other investigators have employed different statistical methods and obtained similar results (Sartorius et al., 1970; WHO, 1973, in Wing et al., 1974).

Wing et al. (1974) note that, on the whole, items covering depressive symptoms have proved to be consistently very reliable while items on anxiety are less so with other symptoms variable and intermediate.

While this study has not used the PSE as a diagnostic instrument on its own, it is worthwhile considering briefly another point in its favour, that is, the reliability of the section and syndrome scores.

Section scores, which are the sum of the scores for the items within each section (e.g. anxiety or depression items), also have a high inter-observer reliability expressed as a product-moment coefficient. For instance, a range of 0.80 and 0.95 was achieved in two studies (Kendell et al., 1968; IPSS), with the section relating to situational anxiety having the lowest correlation coefficient at 0.58. Wing et al. (1974) point out the flaw and the usefulness of this data: 'This high reliability might perhaps be expected, since two observers might arrive at the same section scores, even though the ratings of the constituent items in that section were not the same. In spite of this, however, it is a very important level of reliability to study since the section scores, representing a summary of judgements over a homogeneous group of items, is probably a close approximation to the usual unstandardised judgements about severity of anxiety, depression, tension, etc. that are made in everyday work' (P. 61).

Syndrome scores can also be obtained by taking the sums of the scores for symptoms of a similar kind. For example, syndrome I (nuclear syndrome) is composed of symptoms regarded by Schneider (1959, 1971, in Wing et al., 1974) as 'first rank', in the sense that they are likely to be diagnostic of schizophrenia in the absence of organic features. These symptoms are differentiated from those in syndrome 13 (auditory hallucinations) because in this case the hallucinations are meant to be voices talking to the patient rather than about him. According to Wing et al., 'If this symptom is well enough defined (so that affectively or sub-culturally based hallucinations can be excluded), it is probably as indicative of schizophrenia as any in syndrome I (nuclear syndrome), but it is kept separate until the demonstration is made that it can be specifically rated' (P. 41).

The IPSS material was used to test the reliability of the syndrome scores of the PSE. Two psychiatrists (one interviewer and one observer) rated 190 interviews and another 51 subjects were interviewed twice. On the basis of the first 35 syndrome scores (there are 38 altogether), the product-moment correlation coefficients of reliability fell within the same range as those of the studies mentioned earlier (though the median value is reported to have been higher (Wing et al.,1974).

In addition to having high reliability with respect to item, section, and syndrome scores, it is possible to obtain a high degree of agreement between psychiatrists on a number of diagnostic categories using PSE information alone. Moreover, studies have shown that when the PSE is repeated by another interviewer within a day or so of the first interview, the reliability stays reasonably high, though the

values are somewhat lower (Kendell et al., 1968; Wing et al., 1967; IPSS). For example, Kendell et al. (1968, in Wing et al. 1974) found that, for items, the statistic KAPPA decreased from a mean of 0.71 for inter-observer reliability to 0.41 for repeatability. Similarly for section scores, the values dropped from a mean of 0.84 to a mean of 0.64.

These results indicate that even when there are changes in the patient's symptoms over time and in the person conducting the interview, the PSE, if used properly, is a sound instrument for assessing the current psychiatric status of an individual.

The developers of the PSE have pointed to some difficulties with their data: 'Most of the results refer to the situation in which two psychiatrists (often two who knew each other quite well) are rating one interview. The observer can pick up clues as to how the examiner proposes to rate and the reliability can thus be spuriously high' (Wing et al., 1974, P. 130). Indeed, most of the psychiatrists who participated in the studies had been educated in the same clinical school. Thus, in spite of the fact that the PSE was used with high reliability within research centres, there is the possibility, again acknowledged by the developers, of differences in the way symptoms were understood and rated across the research sites. Wing et al. report instances where 'key symptoms had been rated present although the examples written down at the time did not seem to warrant a positive rating. It is not known how often this occurred without being noted or how often a symptom was present but not rated positively' (P. 130).

The reasons for such differences include: 1) the lack of prolonged formal training in the original group of psychiatrists who conducted

and took part in the early studies; 2) because there was no detailed glossary of definitions of items in the earlier editions of the PSE, training in the differential definition of symptoms was less than adequate; and 3) it is likely that over a period of time psychiatrists with different initial training and ideas gradually deviated from one another.

It is clear from the reasons outlined above that to ensure comparability of results across investigations raters should be trained before using the Present State Examination if this is possible. But what happens when a group of untrained researchers select the PSE as their instrument of choice? Can they expect a reasonably high level of consistency? Recent evidence from Canberra, Australia, indicate that this is possible. Duncan-Jones et al. (1980) found that in a population survey a group of psychiatrists and clinical psychologists can achieve consistency on a number of PSE measures after lengthy self-training and 'regular recalibration' sessions throughout the period of the study. A finding particularly relevant to the present study is that the two psychologists,as a pair, rated at the same level as the psychiatrists. This contrasts sharply with an earlier report (Wing et al., 1977) which suggested that clinical psychologists tend to rate twice as much morbidity as psychiatrists when using the PSE in a population survey.

The main problem with the study is that the Canberra raters might be able to obtain high reliability between themselves but would not get the same consistency against a rater trained by the developers of the PSE or one of their delegates. Even so, the findings should serve to encourage researchers without formal training to use the

PSE in investigations where a systematic description of psychiatric symptoms is desirable.

In summary, the PSE is a very reliable instrument for describing patients who present with psychiatric problems. Although the PSE has many uses it was used in this study primarily to elicit symptoms in a systematic manner to fulfill Spitzer's Diagnostic Criteria for Primary Major Depression. In addition, a total severity score based on PSE items was taken as a measure of general psychiatric disturbance.

(ii) The Research Diagnostic Criteria

The previous section described the Present State Examination, a uniform and reliable procedure for collecting the information used to make diagnoses and to describe the characteristics of patients who participate in psychiatric research. In devising the PSE, Wing and his associates have made a significant contribution to resolving one problem that has plagued investigators in the study of affective disorders.

Another and equally important problem has been tackled by another group of researchers over the past decade, namely, the development of agreed upon sets of criteria for diagnosis which enable researchers to compare the results of studies which use different ways of classifying patients (Feighner et al., 1972; Spitzer et al., 1978). The absence of a uniform set of diagnostic criteria makes it virtually impossible to know if one researcher's patients who are characterised, for example, as having 'severe endogenous depression' correspond to another researcher's patients described as having 'psychotic depression'. Although Wing and his colleagues have also been concerned with this

problem, most of the work on the development of diagnostic criteria for the affective disorders has come out of the United States.

Before talking about the Research Diagnostic Criteria, it would be helpful to shift the focus of the discussion to some important issues related to the contemporary approach to the diagnosis of depression.

A recurrent theme throughout this thesis has been that the depressions represent heterogeneous disorders (Mendels, 1975; Klerman, 1974). However, clinically relevant and commonly agreed upon distinctions between the sub-groups of depressions is still far from adequate. To date, no generally accepted scheme for subdividing the depressions has been found, though numerous investigators have tried to distinguish between the various subgroups (e.g. Garside et al., 1971; Robins et al., 1972; Paykel, 1971; Klerman, 1971).

The modern day approach to the diagnosis of depression stems from the emergence of scientific medicine in the late 18th and early 19th centuries. According to Rush (1976) 'The approach of Sydenhamm in the 18th century led to the classification of diseases by symptoms and clinical course. The development of clinical pathology with clinical-pathological correlates . . . and the development of bacteriology . . . accordingly led to an acceptance of the aetiological principle as the basis for nosology. Thus, the principle that, ideally, classification should be based on knowledge of aetiological processes was accepted' (P. 51).

Kraepelin applied these basic concepts in psychiatry. On the basis of prognosis, he differentiated the depressions (then manic-depressive illness) from the schizophrenias. The concept of 'affective disorders' was developed by Bleuler to incorporate not only manic-

depressive illness but also psychoneurotic depressive reactions, involutional melancholia, etc. Thus, he widened the concept of the depressions, but did not specify the boundaries of the 'affective disorders' - a problem which persists today (Rush, ibid).

Meanwhile, in America the philosophical and scientific bases of psychiatric nosology was being criticised by such eminent figures as Meyer and Menninger. The disciples of Meyer regarded depression as part of the range of human experience, a total reaction to the stresses of life. These and other 'unifiers' such as Aubrey Lewis underscored the importance of social factors and personal experience in the origins of depression and opposed proposals that the depressions should be subdivided. The Meyerians paid less attention to the influence of organic, constitutional and genetic factors in the development of depression. While they accepted the concept of a gradualist or continuum model of depression from minor to major disturbance, they doubted the validity of the distinction between neurotic and psychotic or endogenous and reactive depressions (Klerman, 1974, in Rush, 1976).

Gillespie (1929) was the first to propose the distinction of endogenous and reactive types of depression. This dualistic approach was meant to differentiate depressions which arose in response to environmental stress (reactive) and those without a clear precipitant (endogenous). Such a heterogeneous or pluralistic approach has been embraced by many continental Europeans and some British authors (Klerman, 1974, in Rush, 1976).

Another diagnostic dichotomy is that of 'neurotic' and 'psychotic' depressions. In the United Kingdom this dualism refers to the evidence for a discontinuity with reality as manifested by hallucinations,

delusions, ideas of reference, etc. In contrast, psychiatrists in America see psychotic depression in terms of the severity of symptoms, extent of functional impairment, and even the degree of ego regression, a viewpoint grounded in the work of Fenichel (see Chapter 3). With regard to this distinction, Rush comments: 'To some, there is an implicit assumption in the neurotic-psychotic dichotomy that psychotic disorders are biological, whereas neurotic disorders are due to stress or personality dynamics. Unfortunately, neurotic-psychotic is sometimes confused with the endogenous-reactive dichotomy, although psychosis is not one of the necessary criteria for the endogenous depressive type' (Rush, 1976, P.52).

The problem of differentiating the various subgroups of depressions is complicated by the existence of other dichotomies such as unipolar-bipolar, retarded-agitated, and primary-secondary depressions, all of which have been discussed in depth by various authors (e.g. Akiskal and McKinney, 1975 ; Kendell, 1976; Becker, 1974, 1977).

A recent distinction and the one most relevant to the selection of patients for this investigation is the primary-secondary dichotomy which has been put forward by the St. Louis group (Robins and Guze, 1972; Feighner et al., 1972). Primary affective disorder refers to a disorder in a patient who has previously been well or whose only previous episodes of psychiatric illness consisted of mania or depression. Thus, secondary affective disorders occur in a mentally ill patient who had previously had another psychiatric illness. The distinction is logically superordinate to the unipolar bipolar category (for detailed discussion see Rush, 1976). The main advantage of the primary-secondary distinction is that it avoids two diagnostic dilemmas:

(1) the question of whether a patient is 'neurotic' or 'psychotic' and (2) whether the depression was precipitated by an environmental stress, i.e. the reactive-endogenous controversy. As suggested earlier, while the term neurotic depression is used frequently in clinical practice to denote a supposedly homogeneous group of depressed patients, there is little, if any, evidence to support this contention. Rather, it seems that 'neurotic depression' is an umbrella label for diagnostically and prognistically heterogeneous groups of depressed patients (Akiskal et al., 1978).

This brief overview of the problem of diagnosis in depression indicates that, in spite of general agreement over the presence of a common core of depression, there is wide disagreement over the precise nature of depression and exactly how to classify it. At the risk of putting too fine a point on the issue, a reference to Kendell's (1976) paper may give an idea of the state of confusion in the area. He points out that the 9th edition of the International Classification of Diseases lists depression in 13 categories, not, in his opinion, because there are in reality so many types of depression, but because different countries see depression and its constituents in markedly varying ways. Until the recent attempt to develop uniform diagnostic criteria, along with reliable procedures for describing symptoms, the field of psychiatry was left with the problem of different authors having varying concepts of depression and a situation where numerous categories were needed to accommodate the different ideas of clinicians in over 50 countries.

The Research Diagnostic Criteria (Spitzer et al., 1975, 1978) were developed to help researchers in psychiatry to overcome the

'contemporary confusion', as Kendell puts it, in the diagnostic system. A major purpose of the Research Diagnostic Criteria (RDC) is to enable investigators to select relatively homogeneous groups of patients who meet specified diagnostic criteria. The RDC were developed as part of a collaborative project on the psychobiology of the depressive disorders sponsored by the National Institute of Mental Health. Although the RDC includes criteria for non-affective disorders, its primary focus is on affective illness.

For each functional psychiatric disorder within the RDC there are both inclusion and exclusion criteria. The criteria for Primary Major Depression were outlined in a previous section. Since one aim of this research was to select patients on the basis of symptoms and previous psychiatric history, subjects were chosen according to the Primary-Secondary distinction, thereby avoiding the difficulties posed by the reactive-endogenous and neurotic-psychotic dichotomies. The RDC also provides for subdividing patients into subtypes of depression (e.g. simple maj or and situational major depression) but this was not considered relevant to the research.

In general, the RDC criteria refer to symptoms, signs, duration or cause of illness as levels of severity of impairment. For some of the diagnoses certain symptoms or symptom patterns are of diagnostic importance only if they persist beyond a certain stated duration. Diagnostic terms are frequently defined in the criteria themselves so as to avoid ambiguity as to essential clinical characteristics.

Spitzer et al. discuss the rationale for selecting the specific criteria: 'an attempt was made to operationally define the category in a manner that would achieve maximal acceptance among clinicians

and researchers who use that particular concept. In many cases the criteria are based on good research evidence indicating that the criteria chosen have considerable usefulness for such diagnostic purposes as predicting outcome, response to treatment and familial association. On the other hand, many of the criteria represent an attempt to operationalise concepts whose importance for diagnosis is based primarily on clinical experience rather than the results of formal research studies' (1978, P. 774).

This comment by the developers of the RDC points to one problem in using any diagnostic criteria no matter how well-conceived it is, namely, that of validity.

For example, Nelson et al. (1978) have commented on their experience with the RDC for the diagnosis of primary major depressive disorder. They draw attention to the problem of false positive diagnoses which they attribute to their use of the criteria exactly as written. If the RDC are used in a 'simple checklist manner' a clearly heterogeneous group of patients is obtained. Nelson et al. note that the reason for this could be because earlier versions of the RDC combined criteria for endogenous and neurotic depressions (Woodruff et al., 1974), a point acknowledged by the St. Louis group (Feighner et al., 1972).

Recent evidence suggests that this warning on the validity of the RDC is well-founded. Feinberg et al. (1979) concur with Nelson et al., emphasising that the RDC may be similarly flawed for the endogenous subtype of depression. They compared the RDC for endogenous major depressive disorder with their own clinical diagnoses in 48 consecutive outpatients who presented to their clinic complaining of depressed mood. All of the patients were screened by a psychiatrist in

an open clinical interview and by a social worker who administered the Schedule for Affective Disorders and Schizophrenia (SADS), a structured interview specially designed for the RDC. The clinical diagnoses were revised using follow-up information gathered six months to two years later. In the words of the authors, 'follow up diagnoses were based on response to treatment, other aspects of clinical course and information from the patient's current therapist' (P. 267).

Feinberg et al. report that the RDC resulted in both false-positive and false-negative errors, with 30% of the patients classified incorrectly. There was poor agreement overall between the clinical diagnoses and RDC, the Kappa coefficient being .41. Particularly interesting was the finding that of the six patients incorrectly classified as not endogenously depressed by the RDC, three were bipolar, 'with unequivocal episodes of hypomania meeting RDC criteria'. With regard to the false-positives, eight patients had 'atypical dysphoric presentations' whose symptom pictures were rather different from those commonly recognised as endogenous depression.

Like Nelson et al. (1978), the authors concluded that refinements are needed in the RDC if homogeneous groups of patients can be expected to be selected for research in the affective disorders. Even with improvements they stress the importance of tempering the RDC with clinical judgement.

As seen, information for the RDC is collected by means of direct examination of the patient, i.e. conducting a structured interview (SADS). If the researcher is interested in obtaining a lifetime prevalence of diagnoses, then a lifetime version of the SADS may be used. The SADS interview can be used for diagnosing current episodes

of illness as well as previous episodes and for making multiple diagnoses of functional psychiatric illnesses. Built into the SADS and RDC is a system which gives the researcher an option of examining the data by choosing patients on the basis of degrees of certainty of diagnosis. For instance, there are specific criteria for probable vs definite, such as 'probable' requiring only three items endorsed in an index and 'definite' requiring four or more (for a detailed account see Spitzer et al.'s third edition of the RDC, 1978). Patients selected for this study had to meet a 'definite' diagnosis of primary major depression.

Since the SADS interview was not employed in this present study to elicit symptoms, there is little point in discussing the instrument in detail. It is worth noting, in passing, that the reliability of the RDC categories based on the SADS scale has been tested in three studies, all of which were conducted in the United States. The reliability figures as expressed in Kappa coefficients of agreement were high, ranging from .88 to .90 for Major Depressive Disorder, and .78 to .86 for Primary Major Depressive Disorder (Spitzer et al., 1978). On the surface it would appear that these figures are comparable to those reported for the PSE section and syndrome scores. This, in itself, suggests that the SADS interview might be a better instrument to use for eliciting symptoms if an investigator elects to use the RDC.

However, compared to the PSE, the SADS scale is problematic from the standpoint of cross-cultural testing. The PSE achieves high reliability through extensive use at multiple centres in the United Kingdom and the United States, not to mention nine centres in Eastern Europe. For this reason the PSE was chosen as the standard measure

for eliciting symptoms in the present study, rather than the culturally restricted SADS scale.

However, there is a problem in using the Present State Examination as a screening measure to fulfill the RDC. Although it is comprehensive and reliable, the PSE does not provide all of the information required to meet some of the inclusion criteria for Primary Major Depression. For instance, according to Spitzer et al. (1978), appetitive disturbance may involve overeating and weight gain as well as eating too little, with accompanying weight loss. Similarly, a patient may have sleep disturbance if he reports sleeping too much, even though the conventional view is that sleep disturbance should be regarded as a symptom of depression only when the patient complains of sleep loss, especially in the early hours of the morning. In short, there are no items in the PSE which allow for ratings on symptoms of excessive eating (and weight gain). Excessive sleeping is rated as a dissociative symptom (item 100), this item not being a compulsory one for the interviewer to ask. Unless the PSE is modified specifically to include items which cover these criteria, it is unlikely that the patient will report the symptom during the course of the interview.

Since an unmodified version of the PSE was used in the present study, it is recognised that some patients who might have been admitted to the study on the basis of strict adherence to the criteria for Primary Major Depression were, in fact, excluded because of the limitations mentioned above. On the other hand, the fact that the interviewers were aware of the limitations in the PSE prompted them to make the necessary enquiries, albeit in a clinical and less structured fashion. It was decided that using the RDC in this way would not present any more of a

threat to the validity of the diagnostic criteria than already appears to exist. Moreover, it was felt that even if some patients were incorrectly excluded from the trial because of the limitations in the interview method, such an error would bias the selection in a manner consistent with diagnostic practices in Britain. In effect, the end result would be to minimise the number of patients admitted to the trial who presented with 'atypical symptom profiles', thereby reducing false-positive diagnoses.

Despite these difficulties, Spitzer's Research Diagnostic Criteria provide a much needed partial answer to the problems of the unreliability of clinical diagnostic practices. The use of the RDC makes the results of this research relatively comparable and repeatable for other investigators.

2. Self-report measures of mood

(i) Beck Depression Inventory

The Beck Depression Inventory (BDI) is a self-rating scale which is derived from Beck's cognitive theory of depression (see Appendix V). It was constructed by selecting items that discriminated between depressed and non depressed psychiatric patients (Beck, 1967). It consists of a list of 31 descriptive symptoms referring to characteristic aspects of depression, e.g. pessimism, social withdrawal, suicidal wishes, a sense of failure and physical symptoms. For each symptom there are four or five statements in the first person ranging from a mild or neutral statement to one indicating a severe form of that particular symptom. Each statement is assigned a score of 0, 1, 2 or 3 to indicate the degree of severity of the symptom. The maximum

score on the BDI is 62.

There is evidence for the construct and concurrent validity of the BDI. A central aspect of Beck's theory is that depressed people regard themselves, their world, and their future in a negative way. The model receives support from findings that this 'negative cognitive set' is manifested in the dreams of depressed patients, in their response in certain projective tests and in their self concept. A significant relationship has been found between self-reported depression on the BDI and masochistic dreams (Beck and Ward, 1961, in Beck, 1967), scores on a self-concept test, with high scores indicating a negative concept (Beck and Stein, 1960), the tendency for patients to see themselves as failures in response to pictorial stimuli (Beck, 1961), childhood bereavement (Beck et al., 1963, in Beck, 1967), the tendency to make extremely pessimistic predictions about future performance following inefficient task performance (Loeb et al., 1964) and the tendency to underestimate successful performance (Loeb et al., 1966, in Beck, 1967).

Its concurrent validity, as assessed by the degree of concordance with physicians' ratings has been found to be 0.65 to 0.67 with an American population (Beck, 1961) and 0.61 with a British population (Metcalfe and Goldman, 1965). In addition, the BDI correlates highly with other self-rating scales (Little and McPhail, 1973).

Beck (1961) reports that the reliability of the scale has been found to range from 0.86 to 0.93. With regard to concordance rates between the BDI and observer rating scales such as the Hamilton Rating Scale (HRS), correlations of 0.75 (Schwab et al., 1967) and 0.82 (Williams et al., 1972) have been reported. However, other investigators have noted that these correlations were highest near recovery and lowest

at the acute stage of the depressive episode (e.g. Carrol et al., 1973; Paykel et al., 1973).

Possible explanations for this finding are: the distorting effects of illness on self-perception, lack of insight, and response sets. It appears that the BDI may not be so reliable in quantifying depression as a physicians's rating when the patient is ill (Prusoff, 1972) and this feature may be positively related to the extent of psychopathology present in the patient (Donovan and O'Leary, 1976). Thus, observer rating scores (e.g. HRS) may be better predictors of BDI scores in the future (when the patient is nearer recovery) than at the same time and they may also be a better predictor of future BDI scores than BDI scores are of future observer rating scores.

For the reasons outlined above, and because of the availability of normative data from a British population, the BDI was chosen as the main instrument for assessing self-reported depression in this treatment study.

(ii) Irritability, depression and anxiety scale

The irritability, depression and anxiety (IDA) scale is a self-report questionnaire for the measurement of irritability for use in a clinical context (Snaith et al., 1978). Interest in the association between irritability and psychiatric disorder (e.g. Gottechalk et al., 1963; Buss and Durkee , 1957; Caine et al., 1967), together with the growing awareness of the possible effect of psychotropic medication in modifying the degree of irritability, indicates that a valid measure of this state is relevant to an outcome study such as this.

The IDA consists of a list of 18 descriptive items which refer to three aspects of mood - irritability, depression and anxiety, e.g.

intolerance, poorly controlled anger, level of interest in the environment, autonomic symptoms, etc. Within the IDA there are four subscales. There are five items for the depression scale (rangeof scores 0 - 15), five for anxiety (range 0 - 15), four items for outward directed irritability (range 0 - 12) and four for the inward directed irritability scale (range 0 - 12). A fifth score can be obtained for total irritability by adding the outward and inward irritability scores. Each item is followed by four possible responses. The wording of some items expresses a healthy state, e.g. 'I feel cheerful' while others convey a morbid state, e.g. 'I lose my temper and snap at others'. The wording of the response format varies from one item to another. On the questionnaire a depression item, an anxiety item and an irritability item appear in sequence. The subject is asked to underline the response which applies most to him/her at the time. While the scoring values do not appear on the questionnaire, scores of 0, 1, 2 or 3 are given depending on the severity of the symptom (see Appendix VI). All of the items on the scale refer to the patient's present mental state, the time period being how the patient feels at the moment or has felt in the previous day or two.

It was decided that a scale which combined measures of irritability with anxiety and depression ratings would be useful because it might help to disentangle the complex relationships between these moods. Another reason, pointed out by the authors of this scale, is that by interspersing irritability items among other items relating to mood disorder, the scale is less likely to put respondents 'on the defensive', thereby increasing the likelihood that statements would be truthfully endorsed.

The scale is relatively new and has not received much empirical

support except for that provided by the developers of the questionnaire.

The correlations of the self-assessment scales with modified versions of the Hamilton Rating Scale for Anxiety and Depression are as follows: depression = .75, anxiety = .70, outward irritability = .79, and inward irritability = .84. These are all highly significant and satisfy the requirements for concurrent validity.

In terms of the sensitivity and specificity of each of the sub-scales, Snaith et al. compared the responses of 78 patients with those of a non-patient control group (N = 100) and found that the depression, anxiety and irritability scales provided satisfactory discrimination between the two groups. For the depression scale 8% of controls and 14% of patients were misclassified according to a psychiatrist's rating of severity of disorder; for anxiety the figures were similar while the most sensitive scale was inward irritability, where only 4% of controls and 8% of patients were misclassified. The least sensitive scale appears to be outward irritability as the percentages of mis-classification were considerably higher, i.e. 16% for controls and 24% for patients.

Snaith et al. checked the reliability of the IDA by internal consistency, dividing the subscales into two halves. While there are difficulties in using this method on scales with few items, the authors found the reliability coefficients (Spearman-Brown method) to be high. Because the scales involved as few as four items, the authors split each scale three ways and obtained the following correlations: depression = .74, .80, .87; outward irritability = .77, .80, .88; and inward irritability = .70, .92, .93.

This scale was chosen mainly because it is one of the few self-report measures available for assessing a subject's present state of

irritability. Although the scale is new and in need of further investigation, its construction was purposefully based on both inpatients and outpatients which makes it valid for use with either population. Moreover, it is a short and convenient way to obtain more information about the complex relationships between several dysthymic states which a patient may experience during the course of a depressive illness.

3. Self-report measures of cognitive content

(i) Semantic differential

This is an instrument for measuring a person's cognitive structuring or the meaning which he/she gives to various aspects of the world. The technique described by Osgood et al. (1957) was modified to provide an assessment of the patient's cognitive world by means of twelve seven-point scales applied to five constructs considered important in depression (see Appendix VII). The bipolar adjectives which occupied each end of the 12 scales represent a selection from those recommended by Osgood et al. (op. cit.) and were chosen for their relevance to depressed mood which had been established in a study in the Metabolic Unit, Thomas Clouston Clinic (Ludgate, 1976). The constructs included Beck's (1967, 1976) negative cognitive triad, i.e. the self, the world and the future. The five constructs were: Me as I am now, Me as I was before this illness, Me as I would like to be, My environment as it is now, and The future as I see it for me. These concepts were rated on the following bipolar descriptors, most of which were derived from Osgood's evaluative factor: successful-unsuccessful, good-bad, worthwhile-worthless, hopeful-hopeless, active-passive, interesting-dull, fast-slow, pleasant-unpleasant, positive-negative, in control-helpless, adequate-inadequate and happy-sad. Depending on

where the patient placed his mark on the line, he was assigned a score of 1, 2, 3, 4, 5, 6 or 7, the lower the score the more negative the rating, the higher the score the more positive the rating. The lowest score a patient could get for a given concept was 12 while the maximum score was 84.

The estimation of reliability for 'state' scales by the test-retest method is, on the whole, unsatisfactory. If the scale is given again to the patient too soon, scores may be affected by recollection of the previous completion. If the time interval is longer the patient's clinical state may have changed.

Bearing in mind these limits, data on the reliability of the semantic differential as derived from test-retest analyses may be considered. In the two studies reported below reliability was assessed in terms of the error of measurement between tests, the finer or smaller the average magnitude of this error the more reliable the instrument.

Luria (1953, in Osgood et al., 1957) used the semantic differential in a psychotherapy investigation. The reliabilities of 150 items (15 concepts and 10 scales) were measured over test-retest intervals of a few minutes, i.e. immediate test-retest, 6-8 weeks, and 12-15 weeks for non therapy controls, and over only the few minute interval for therapy patients. Error was assessed in terms of the 'average absolute deviations in response from test-retest'.(P.131), for all scales, and the three categories of scales. The categories consisted of the three factors - evaluation, potency and activity (EPA) - which emerged from Osgood et al's factor analysis of over 50 bipolar adjective scales across 20 different concepts (see Osgood et al., 1957 for a detailed account). Looking at the immediate test-retest data for the non-therapy controls and therapy patients, evaluative scales (e.g. worthwhile-worthless)

produced the smallest average errors, these being .53 for the controls and .58 for the therapy group. This contrasts sharply with the activity scale (e.g. active-passive) which yielded the largest error measurements: .85 for the non-therapy controls and .81 for the therapy patients. There was no difference in reliability between normal controls and 'neurotic' patients but, as expected, the magnitude of the average error increases considerably with longer time intervals between tests.

Test-retest reliability of the somantic differential has also been examined in a study by Bapp (1955, in Osgood et al., 1957) in which groups of normal controls and schizophrenic patients were compared in immediate and delayed (two weeks) testing conditions. Again, the evaluative scales showed consistently smaller deviations for both test-retest intervals and for both groups than the potency and activity scales. However, the schizophrenic patients, in contrast to Luria's patients undergoing psychotherapy, showed significantly poorer reliability than the controls ($P = 0.001$ by Mann-Whitney test). It may be that this finding points to the limitations of the semantic differential when applied to schizophrenics, or perhaps less stability in their meanings of concepts (i.e. change in what is being measured).

As a measure of general attitudes, the semantic differential achieves high test-retest reliability. For example, Tannenbaum (1953, in Osgood et al., 1957) looked at each of six concepts judged against six evaluative scales. One hundred and thirty-five subjects were seen on two occasions separated by five weeks. The test-retest coefficients ranged from .87 to .93 with a mean r (Z transformation) of .91.

Unfortunately, studies of the validity of the semantic differential are almost exclusively concerned with the evaluative factor. For

instance, Suci (1952, in Osgood et al., 1957) was able to discriminate between high and low ethnocentrics , as determined separately from the E scale of the Authoritarian Personality studies, on the basis of their ratings of different ethnic constructs on the evaluative scales of the semantic differential. Similarly, it was found that the evaluative scale ratings discriminated in their predicted ways between subtleties in political preferences (Luci et al., 1952) and pictorial political symbolism (Tannenbaum and Kessick, in Osgood et al., 1957).

Its concurrent validity as assessed by the degree of concordance with attitudes rated on a Guttman scale is reported to be high (.78, $P < 0.01$, being the rank order correlation between the two instruments by test-retest method).

Melges et al. (1971) used the semantic differential to determine the relationship between self esteem and perception of personal future. By correlating semantic differential scores for self esteem and future outlook from normals and psychiatric patients and by correlating changes in self-esteem with changes in future outlook within acutely ill patients over time, they found that the degree of a person's self-esteem is significantly related to how hopeful he is about his own future. The authors speculated that therapies (e.g. cognitive therapy) which offer a specific approach to modifying future outlook should be helpful in treating patients with low self-esteem.

Self-concept has been demonstrated to improve after treatment in depression (Laxer , 1964). Depressives often initially show high self-blame and low self-ratings which is probably a function of mood level. There is a change towards lower self-blame and higher self-

ratings after treatment (Laxer, ibid). In addition to the enhancement of the self-concept it would be expected that constructs like 'the environment' (world) and 'the future' should also change positively as depression lifts. More importantly, if Beck's cognitive model of depression is to be empirically supported, then these cognitive changes should precede mood changes in the course of recovery.

In summary, the semantic differential can be applied to a variety of research problems. It has been shown to be sufficiently reliable and valid for many research purposes. It is also flexible and relatively easy to adapt to varying research demands, quick and economical to administer and to score. And, as Heise points out, 'there is probably no social psychological principle that has received such resounding cross-group and cross-cultural verification as the EPA structure of the SD ratings' (1965, P.421). The main problems are to select appropriate and relevant concepts and descriptive scales as well as appropriate and relevant analysis. In this study the concepts were relevant to Beck's cognitive model and the descriptive scales and the instrument was used as an outcome measure to assess changes in cognitive aspects of depression.

(ii) General expectancy scale (hopelessness scale)

In recent years considerable work has been directed at the importance of hopelessness in a variety of psychopathological conditions. Thus, hopelessness has been named as one of the central features of depression (Beck, 1963, 1967, 1973; Melgis and Bowlby, 1969) and has been implicated in a number of other conditions such as suicide (Beck, 1963), schizophrenia (Laing and Esterson, 1965), alcoholism (Smart,

1968), psychopathy (Melges and Bowlby, 1969) and physical illness (Schmale, 1958).

The hopelessness scale (HS) can be seen as a self-rating scale designed to measure pessimism, or an individual's negative expectations about his personal future (see Appendix VIII). It consists of 20 items which are rated true or false, 9 of which are keyed 'false' and 11 keyed 'true'. For every statement, each response is assigned a score of 0 or 1 and the total hopelessness score is the sum of the scores on the individual items. Thus, the possible range of scores is from 0 to 20.

Published data on the psychometric properties of the hopelessness scale are scarce. However, Beck et al. (1974) have reported on the reliability and validity of the scale. They checked the reliability of the HS by looking at its internal consistency in a sample of 294 patients who had been hospitalised for attempting suicide. The analysis produced a reliability coefficient of .93 (ALPHA coefficient). In terms of scale intercorrelations, the item total coefficients were reported to range from .39 to .76.

The degree of concordance with doctors' ratings of hopelessness has been found to be .74 in a general medical practice population (N = 23) and .62 with a hospitalised attempted suicide sample (N = 62). Comparing the HS with other measures of hopelessness, Beck et al. (1974) found correlations of .60 with the Stuart Future Test (Stuart, 1962) and .63 with the pessimism item of the Beck Depression Inventory, this being the highest item correlation on the BDI.

The construct validity of the HS, as assessed through factor analysis (N = 294) is open to question. Although the scale is meant

to tap a person's 'negative expectations of the future', factor 1, which revolved around affective associations (e.g. happiness, enthusiasm), accounted for the largest amount of variance (42%). On the other hand, only 6% of the total variance was accounted for by factor 3 ('future expetations') which dealt with the individual's anticipations about what life would be like, the likelihood of things working out, etc. This would help to explain the significantly high correlation (Beck et al., 1974) between the HS and the BDI (.63, $P < .001$), a self-report measure of depression heavily loaded with affective content, e.g. self-debasement (Pichot and Lemperiere, 1964, in Beck, 1967).

Despite these difficulties, the measurement of pessimism has been useful in exploring possible motives for suicidal behaviour. For instance, Minkoff et al. (1973) reported that the HS correlated markedly higher with suicide intent in suicide attempters ($r = .47$, $P = < .001$) than did severity of depression as measured by the BDI ($r = .26$, $P = < .05$). Further analyses showed that the partial correlation of suicide intent with HS (holding BDI constant) was .41 ($P = < .001$) while partial correlation of intent with BDI (holding HS constant) was -.09 (nonsignificant). These results suggest that hopelessness may be a better predictor of suicide intent than depth of depression.

The relationship between hopelessness, depression, and suicide intent has also been investigated by Wetzel (1976). He examined a sample 154 suicide attempters, suicide ideators (threateners) and psychiatric controls, i.e. psychiatric patients who denied any suicide attempt or ideation within one year of testing. Subjects were rated on suicide intent scales and given the HS, Zung depression scale, and

a self-rated measure of suicide risk. Ninety-four out of the initial sample were administered these scales again one month later. Suicidal subjects were found to be more depressed than the psychiatric controls. Moreover, severity of suicide intent in threateners and attempters was significantly ($P = < .05$) associated with depression.

With regard to hopelessness, HS scores discriminated between suicidal patients. In all cases hopelessness was more highly correlated with suicide intent or behaviour than depression. Like the previous study, when the effects of hopelessness were controlled statistically in ideators, depression no longer correlated with suicide intent. However, when depression was controlled, hopelessness and suicide intent were still significantly correlated ($r = .41$, $P = < .01$).

Although there are problems with the validity data for the HS, it seemed justifiable to use it in this study because of the documented relationship between hopelessness and level of depression. As the HS has not been used in a British sample of depressed patients, it was decided that the validity of the scale should be checked by comparing patient responses on the HS with the other measure of personal future used in the present study, i.e. 'the future as I see it for me' (one of the concepts on the semantic differential).

Clearly, more extensive validity evidence pertaining to psychiatric and nonpsychiatric patients, the base rate, and its sensitivity through repeated clinical use is required for the HS.

4. Observer rating

One of the first rating scales primarily designed for depressive disorder was described in 1960 and revised in 1967 (Hamilton, 1967). The Hamilton Rating Scale for Depression (HRS-D) was designed to be used by those who have adequate clinical experience and have been trained in the use of rating scales (Hamilton, 1974). It has become the standard instrument for psychiatrists' ratings and has been said to have no serious rival as an observer scale (Carrol et al., 1973). It is meant to be completed after a clinical interview and to take account of information from all available sources concerning the patient's behaviour during the previous week. Thus, the HRS-D cannot be used too often in the course of a treatment trial.

The scale consists of 17 items which are added to give a score measuring severity of illness. One common symptom 'diurnal variation' is excluded because it is not a symptom which adds to the severity of the illness but records the form of the illness (Hamilton, 1974). Other relatively rare symptoms not included in the total score are depersonalisation, obsessional symptoms and paranoid symptoms. Each of the 17 items is scored on a three (0 to 2) or five (0 to 4) point scale for its intensity and/or frequency (see Appendix IX).

The reliability has been found to range from 0.81 (Prusoff et al., 1972) to 0.90 (Hamilton, 1979) between raters at the same interview. The validity of the scale as assessed by its correlation with other instruments for measuring depression ranges from 0.79 (Brown and Zung, 1972) to 0.82 (Williams et al., 1972). Zealley and Aitken (1969, in Hamilton, 1979) reported that for patients just admitted to hospital the correlation with global judgements was .90 while for patients who

were discharged it was 0.55. This decrease in reliability, according to Hamilton, may be due to the decreased range in scores.

The HRS-D has been criticised on the grounds that there is too much emphasis on somatic or biological changes (Hamilton, 1979). Apart from this, the main disadvantage with the scale is that it must be administered by trained and experienced raters. The use of skilled interviewers is time consuming and costly.

Given the experience of the raters, the strongest point about the Hamilton Scale is that it is a reliable and valid observer rating instrument. A skilled observer, by reason of his training and clinical expertise, has developed standards against which he can evaluate any one symptom, whereas a patient does not have the clinical background required for a valid assessment. Patients occasionally have their reasons for minimising or over-emphasising their symptoms and a skilled observer may be able to offset these difficulties through a good interview, thereby obtaining a more accurate account of the patient's state (Hamilton, 1979). Additional advantages of the scale are that it is short enough to allow easy completion yet thorough, it permits flexibility in interviewing and is not restricted to the here and now assessment (Hamilton, 1974).

5. Behavioural measures

Slowness of responding is generally considered to be a major symptom of depression and seems to be reversible with improvement in clinical status (Miller, 1975; Blackburn, 1975). For example, Fisher (1949) reported a significant increase in speed on the Digit Symbol Test of the Wais for a group of depressives who had been successfully treated with ECT. Other studies (e.g. Hall and Stride,

(1954) found significant decreases in reaction times for both schizophrenic and endogenous depressed patients. Miller (1975), in his review of the area, notes that more severely disturbed patients (e.g. 'endogenous' or psychotic depressives) perform very slowly on motor tasks while the less severely disturbed patients (e.g. 'neurotics') are, in fact, slow to respond but more like normals. Thus, a useful index of severity of illness appears to be psychomotor retardation.

In the present study behavioural tasks were used to assess baseline retardation and improvement with treatment in an attempt to back up the other verbal report measures with more indirect behaviour measures which, it can be argued, are less sensitive to demand characteristics.

(i) Speech rate

One of the behavioural tasks used in this investigation was based on a technique described by Szabadi et al. (1976). They found that a sample of 'automatic speech', i.e. counting from one to ten, taperecorded and measured for elongation of pauses between phonation differentiated groups of normals from moderately depressed inpatients. Although the pause time of the volunteers remained constant over a two month period, the patients showed a marked decrease in pause time as they recovered from depressive illness. This appeared to represent a real change in their state and was not due to practice effect because no change in pause time had been observed in the healthy volunteers over a similar period of study. Moreover, this simple behavioural measure seemed more sensitive and produced more consistent results than other tests for retardation (Hamilton Retardation Scores, motility scores from nurses' ratings and tapping time).

As the authors recommended that the test could be further simplified

(without losing its sensitivity) by measuring total counting time (i.e. phonation plus pauses), it was decided that this modified version of 'automatic speech' would be used in the present study.

Thus, at each assessment subjects were asked to count from 1 to 10 'taking their own time' and this sample of automatic speech was recorded using a Sony portable tape recorder equipped with a hand microphone. The recording was later played back through a Grass Polygraph and the signals magnified by a control voice box. Once the auditory output had been magnified and printed onto paper (speed = 50 mm/sec.) the total length of the voice trace was measured in hundreths of a second (an example of a 'voice trace' appears in Appendix X).

The results of the periodic speech tests were evaluated by an independent staff member, a psychophysiological technician in the Brain Metabolism Unit, and thus neither the experimenters nor the patients received any feedback about the performance on the test.

The automatic speech test was chosen because of its simplicity, shortness, ease of administration, sensitivity to slowness of response and the fact that it is unlikely to be contaminated by I.Q.

(ii) Writing speed

The other behavioural task used in this study was writing speed. Patients were asked to count backwards to themselves starting from 100 and to write these numbers on a sheet of paper, e.g. 100, 99, 98, 97, 96, etc. The patient was given a time-limit of 90 seconds, though this was never made explicit. The reason for not telling the subject about the time limit was that the examiner wanted to obtain as much

of a 'spontaneous performance' as was feasible within the limitations of the assessment situation. The more numbers written correctly in sequence the higher the score on writing speed. The range of scores was 1 to 100 or more, as some subjects had time to start again from 0 within the 90 seconds.

Speed of performance has been measured using similar psychomotor tasks (Babcock and Levy, 1940; Rapaport, 1945; Fisher, 1949; Foulds, 1952; Shapiro and Nelson, 1955; Payne and Hewlett, 1960; Blackburn, 1975). It was thought that an alternative behavioural task would be useful, especially one that is simple, yet more demanding of attentional processes and motor skills.

H. SUMMARY OF MEASURES USED IN THE STUDY

Table $5H_1$ shows a list of the measures used in the present study. There were two diagnostic instruments, seven mood, nine cognitive and two behavioural measures, making a total of 20 measures. Of these 18 were outcome measures.

Under the column labelled 'cognitive measures' numbers 7, 8 and 9 represent aspects of cognition that were derived from the five concepts used in the semantic differential. In view of the literature relating depression to premorbid personality, and excessively high standards, it made psychological sense to measure what might be termed (1) the patient's perceived deviation from previously 'well' self (no. 7), (2) the perceived degree of discrepancy between ideal self and current self (no. 8), (3) and the perceived difference between ideal self and previously 'well' self (no. 9).

With regard to these 'composite measures', the maximum numerical

Table $5H_1$ Measures used

Diagnostic	Outcome		
	Mood	Cognitive	Behavioural
1. Present State Examination (PSE)	1. Beck Depression Inventory (BDI)	1. Hopelessness Scale (HS)	1. Speech rate
2. Research Diagnostic Criteria (RDC) Primary major depression	Irritability, depression, anxiety (IDA)	Semantic Differential:	2. Writing speed
	Subscales:	2. Me as I am now	
	2. Inward Irritability	3. Me as I was before this illness	
	3. Outward irritability	4. Me as I would like to be	
	4. Total irritability	5. My environment as it is now	
	5. Anxiety	6. The future as I see it for me	
	6. Depression	7. Perceived deviation from previous self, i.e. score on 3 minus score on 2	
	7. Hamilton Rating Scale (HRS)	8. Perceived discrepancy between ideal and current self, i.e. score on 4 minus score on 2	
		9. Perceived discrepancy between ideal self and previously 'well' self, i.e. score on 4 minus score on 3.	

difference between any two concepts is 72, or the difference between the highest possible score for a concept (84) and the lowest possible score (12). Thus, the greater the difference score the greater the discrepancy between, for example, ideal self and estimate of current self.

I. GENERAL HYPOTHESES

1. Patients from the two referral sources will differ in level of depression pattern of depressive symptoms and severity of psychiatric symptoms at presentation for admission into the trial.

The reason for putting forward this hypothesis was to test the supposition held by some hospital-based staff that hospital patients are more likely to report symptoms indicative of depressive illness (syndrome depression) and greater intensity of depressed mood than general practice patients. There is evidence that patients referred for psychiatric treatment are a selected group (e.g. Rawnsley and Loudon, 1962), in that they have chronic disorders, often with attendent social and behavioural problems (Shepherd et al., 1966). Moreover, patients in community-based samples are reported to present with lower levels of general psychiatric disturbance (Wing et al., 1978).

2. The combined cognitive therapy and pharmacotherapy treatment modality will have fewer drop-outs than either of the cognitive therapy and pharmacotherapy groups.

Herceg-Baron et al. (1979), using interpersonal psychotherapy (IPT) in combination with drugs, found such an effect (P. 41). Rush et al. (1977) also found this effect in their study of cognitive therapy alone.

3. Patients assigned to each treatment modality will show a reduction in depressive symptoms by the end of the specified period.

This hypothesis is derived from the findings of numerous drug studies (e.g. Morris and Beck, 1974). A few investigations on the efficacy of cognitive therapy (e.g. Rush et al., 1977; Shaw, 1977) and the recent study by Weissman et al. (1974) concerning the effectiveness of interpersonal psychotherapy in conjunction with drugs which have shown these forms of treatment to be effective in relieving the acute symptoms of depression.

4. Patients who receive cognitive therapy will show a greater reduction in severity of depression than those given pharmacotherapy (P. 140).

5. Patients assigned to the combination of pharmacotherapy plus cognigive therapy will show a greater reduction in acute symptomatology than either of the cognitive therapy or pharmacotherapy groups (P. 40).

6. Patients who respond to the combination treatment will show a quicker reduction in depressive symptoms than responders in either of the cognitive therapy or pharmacotherapy groups.

This hypothesis is related to hypothesis 5 and refers to the presumed positive interactive effect of the two efficacious treatment modes.

J. SPECIFIC HYPOTHESES

Specifically, this research focuses on the following hypotheses:

1. Hospital outpatients will have higher levels of self-reported (BDI) and observed (HRS) depression than general practice patients.

2. Hospital outpatients will report more diagnostically relevant depressive symptoms as measured by the PSE than general practice patients.

3. Hospital outpatients will be more severely disturbed in terms of overall psychiatric symptoms than general practice patients as documented by total score on the PSE.

4. Cognitive therapy will be superior to pharmacotherapy in reducing the number of drop-outs from treatment.

5. Combination treatment will be superior to cognitive therapy and pharmacotherapy in reducing the number of drop-outs from treatment.

6. Patients in all three treatment groups will have lower scores at the end of treatment than they did at the start of treatment on all mood measures, i.e. BDI, the depression subscale of the IDA, anxiety, inward irritability, outward irritability, total irritability (IN + OUT) and the HRS.

7. Patients in all these treatment groups will have lower scores on hopelessness (HS) at the end of treatment than they did initially. Since the semantic differential is meant to measure the degree of negative thinking about the self, the world and the future, patients in the three treatment groups will have higher scores on these concepts at the end of treatment than they did at the beginning. Thus, it is predicted that as patients recover from depression they will view themselves, their environment and their future in a more positive light.

8. Patients in the three treatment groups will perform better at the end of therapy than they did at the beginning on both behavioural tasks, i.e. they will count from 1 to 10 faster (speech rate) and write more numbers in the allotted time at the end of treatment relative to the beginning.

9. Patients assigned to the cognitive therapy group will show greater reductions in mood disturbance on all mood measures at the end of treatment compared to those assigned to pharmacotherapy.

10. Patients receiving cognitive therapy will show a greater reduction in hopelessness and higher scores on views of self, world, and future than patients who receive pharmacotherapy.

11. Those who receive cognitive therapy will also perform better on the two behavioural tasks at the end of treatment compared to the pharmacotherapy group.

12. Patients assigned to the combination of pharmacotherapy and cognitive therapy will show the greatest reduction in acute affective symptoms at the end of treatment on all mood measures.

13. Patients assigned to combination treatment will show the greatest reduction in hopelessness and the greatest increase in scores on views of self, world and future compared to the other treatment groups.

14. Combination therapy will result in the best performance on the two behavioural measures at the end of treatment compared to the other two treatments on their own.

15. Patients who respond to combination treatment will show a more rapid reduction in self-reported depression (BDI) than those who respond to cognitive therapy alone and pharmacotherapy alone.

16. Patients who respond to cognitive therapy and the combination of drugs and cognitive therapy, because of the specific focus of cognitive therapy on hopelessness, will show a more rapid reduction in degree of hopelessness as measured on the hopelessness scale (HS) than those who respond to pharmacotherapy.

K. STATISTICAL ANALYSES

The statistical techniques used in this investigation were all standard procedures. Most of the data for group comparisons were analysed using the Statistical Package for the Social Sciences (SPSS, second edition, 1975) on the EMAS 2980 computer system.

1. Categorical (i.e. nominal scale) comparisons, except for three variables (i.e. referral source, education and social class) were made by means of the chi-square test, or where more appropriate, the Fisher Exact Probability Test (Siegal, 1956).

2. Group comparisons at baseline and for outcome were analysed by two way analysis of variance or covariance for unequal cell sizes (see Winer, 1970; Snedecor and Cochran, 1967; SPSS second edition, 1975) for treatment and location (i.e. hospital and general practice settings).

3. In the analyses of outcome, significant differences as determined by F ratios were followed up by applying Snedecor and Cochran's (6th edition, 1967) revised least significant differences method in order to locate significant differences between means.

4. Other two-group comparisons were calculated by Student's t test for related or independent samples (Guilford, 1965). Even where directional predictions could be made, significance levels reported will be for two-tailed tests, unless otherwise stated.

5. Correlations were calculated using Pearson product moment coefficient (Guilford, 1965; SPSS second edition, 1975).

6. The differential response of endogenous and non-endogenous patients to each treatment mode was examined by X^2 analysis, as were the number of endogenous responders and non-responders, and non-endogenous responders and non-responders in each treatment.

7. Graphs were plotted for self-reported depression (BDI) and hopelessness (HS) to see whether the treatments produced different response patterns.

8. In an attempt to predict which variables best predicted response to each treatment, three multiple regression analyses were computed, one for combination, CBT and pharmacotherapy (see Darlington, 1968; Kerlinguer and Pedhazen, 1973, for a detailed account of multiple regression).

9. In a rather hypothetical extension to the multiple regression techniques, patients from the treatment groups were re-assigned to three 'predicted treatment groups' on the basis of individual characteristics which had been identified as important predictors of response to combination treatment, CBT and drugs.

A discriminant function analysis was then computed to elicit further psychological factors which might predict response to treatment. This, however, was a highly abstract exercise, and was intended largely to raise some questions for future research.

A complete description of the rationale and results of the analysis appears in appendix XIX (for a detailed description of discriminant function analyses see Tatsauka, 1970, 1971; Rao, 1978; and the Manual for the Statistical Package of the Social Sciences, 2nd edition, 1975).

It is not proposed to specify here the statistical techniques used to investigate each particular area of interest in this research. The different stages of the data analysis went in sequence and the details of the method of analysis at each stage were determined to some extent by the results at preceding stages. Therefore, it was decided that it would be more meaningful to describe the relevant statistical methods briefly at the beginning of each section of the results chapter.

CHAPTER SIX

RESULTS

A. DESCRIPTION OF THE SAMPLE

The Present State Examination is a valuable aid for describing the psychopathological characteristics of groups of people at a specific point in time. Since patients were referred from two sources, a hospital and a community-based clinic, and because the PSE could be expected to reveal any relevant differences between these groups, it was considered desirable to compare these groups in terms of symptoms obtained from the PSE. As the PSE was used to fulfill the RDC for Primary Major Depression, X^2 analyses (2 x 2 tables) were computed only on diagnostically relevant items. The decision to use the X^2 test in these and other comparisons was based strictly on Siegel's (1956) recommendations for X^2. Two tailed tests of significance were used in all instances.

A complete symptomatic description of the total sample from each referral source appears in Appendix XI.

It was anticipated that these groups would also differ on several clinical, demographic, and severity measures. Therefore, a 2-Way Analysis of Variance was computed which compared the two populations for each of the three treatments. This analysis permitted inspection of possible differences across treatments and took account of any interaction effects between the separate populations and treatment modalities.

Few problems create greater difficulties in a treatment study than differential attrition. Attrition, by definition, is not under

experimental control. When subjects discontinue treatment for different reasons in the different treatment conditions, the adequacy of random assignment to treatments is undermined. Observed differences between groups cannot be clearly attributed to the experimental manipulation.

No direct attempt was made in this study to elicit reasons for dropping out of treatment. However, it was decided that it would be helpful to find out in what ways completers could be differentiated from noncompleters in terms of clinical, demographic, and outcome measures. This strategy provided some means of evaluating the extent of bias related to differential attrition. Thus another two-way analysis of variance was computed which compared patients who completed treatment with those who did not (irrespective of referral source) in the three different treatment groups.

The last comparison which was made involved only those patients who completed the trial. A two-way analysis of variance was calculated which compared the final sample from each population within and across the three treatments (i.e. the same analysis that was computed for the entire sample).

Finally, a correlational analysis was computed for the entire sample which was intended to establish the extent to which there was an interrelationship between demographic, clinical and dependent variables. Again, two-tailed tests of significance were employed in the analysis.

1. Comparison of the hospital and general practice patients on symptoms

From table $6A_1$, the different patient samples were significantly related to only two symptoms as elicited by the PSE, loss of libido and subjective anergia, with the hospital outpatient sample reporting these symptoms more frequently than general practice patients. The Research Diagnostic Criteria for Primary Major Depression as derived from the PSE symptoms, including rating criteria, appears in Appendix XI, as does a complete description of PSE symptoms obtained from the patients in each clinic.

Table $6A_1$ Presence or absence of symptoms used to meet the diagnostic criteria in the hospital and general practice sample

		Frequency present Hospital N = 49	Frequency present G.P. N = 38	X^2, df = 1
23.	depressed mood	49	38	Not applicable
19.	inefficient thinking	37	23	1.59
20.	poor concentration	36	28	.07
22.	loss of interest	40	24	2.87
38.	loss of libido	23	9	4.03 *
28.	avoidance of social contact	36	24	.64
35.	delayed sleep	22	22	.97
37.	early wakening	19	21	1.73
34.	loss of weight	18	12	.08
29.	self-depreciation	34	20	1.89
33.	self blame	28	18	.48
25.	suicidal ideas/plans	27	17	.55
6.	loss of energy	43	34	.008
36.	subjective anergia	42	24	4.79 *
26a	anxiety and depression	11	8	.17
26b	depression primary	38	30	.01
110.	slowness/underactivity	4	2	.01
111.	agitation	5	1	.91

* $P = < 0.05$

N.B. in the general practice group N = 38 for PSE data only (1 PSE missing)

2. Comparison of the initial sample of hospital and general practice patients on clinical, demographic and baseline measures of severity

Before investigating the effectiveness of each treatment separately and relative to one another, clinical, demographic, and severity measures of depression in the two populations at the time of admission into the trial were examined. The reasons for this were: (1) to provide basic information on the population which could be compared with other studies, and (2) to find out whether the differences, if any, were sufficiently large to require more sophisticated analysis and further consideration when discussing the outcome data.

The statistical design employed was that of a 2 x 3 analysis of variance with unequal group sizes. This design and the analysis are described by Winer (1970). Anticipating that any significant differences between groups of completers would be controlled statistically in the analysis of change scores, it was considered unnecessary at this stage to do a-posteriori tests of significance or to distinguish interactional effects from simple main effects.

In the summaries of the analyses of variance presented throughout this chapter, the notation for all variables, the two samples, and three treatment modalities is as follows:

Variable	Notation
1. Location	Loc
a) hospital	H.P. (1)
b) general practice	G.P. (2)
2. Treatment	TR
a) Combination	COMB
b) Cognitive-Behaviour Therapy	CBT
c) Drugs	D

3.	Age	AGE
4.	Education	EDUC
	a) level I (lowest)	1
	b) level II	2
	c) level III	3
	d) level IV (highest)	4
5.	Social Class	SOC
	a) I (highest)	1
	b) II	2
	c) III	3
	d) IV	4
	e) V (lowest)	5
6.	Duration of current illness (in weeks)	DILL
7.	Present State Examination (total severity score)	PSE
8.	Beck Depression Inventory	BDI
9.	Hopelessness Scale	HS
10.	Depression (I.D.A.)	DEP
11.	Anxiety (I.D.A.)	ANX
12.	Inward Irritability (I.D.A.)	IN
13.	Outward Irritability (I.D.A.)	OUT
14.	Total Irritability (IN + OUT)	IRR
15.	Hamilton Rating Scale	HRS
16.	Me as I am now	AM (current self)
17.	Me as I was before this illness	WAS
18.	Me as I would like to be	WD (ideal self)
19.	My environment as it is now	ENV

20.	The future as I see it for me	FUT
21.	Perceived deviation from previous self	WASE (WAS minus AM)
22.	Perceived discrepancy between ideal and current self	IDS (WD minus AM)
23.	Perceived discrepancy between ideal self and premorbid self	IDW (WD minus WAS)
24.	Speech rate (Counting Speed)	CS
25.	Writing Speed	WS

Table $6A_2$ summarises the results of the analysis of variance computed for all of the patients admitted into the trial (for F ratios and further information see Appendix XII).

There were five location differences that were statistically significant, indicating that the hospital patients were better educated, from a higher social class, were depressed for a longer period, were more severely disturbed in terms of reported severity of symptoms on the PSE, and had lower self-esteem than the patients seen in general practice. Statistically significant differences between treatment groups were found for three variables. Patients assigned to cognitive therapy and combination treatment were ill for a longer period on average than those assigned to drug treatment. The combination treatment group also had a higher mean HRS score than either the cognitive therapy or pharmacotherapy group. The drug treatment group performed less well on one psychomotor task, as evidenced by a relatively lower mean WS score, than either of the cognitive therapy groups.

There was only one interaction effect. Inspection of the results for duration of illness shows that hospital patients assigned to either of the cognitive therapy treatments had been depressed for a longer

time on average than hospital patients assigned to pharmacotherapy or the three general practice treatment groups. In the general practice sample, the patients allocated to cognitive therapy had experienced the most chronic depression on average, while the least chronic patients were those allocated to the combined cognitive therapy plus pharmacotherapy group.

Thus, on the whole there were only few differences between the samples from the two sources of referral.

Table $6A_2$ Results of analysis of variance on all patients admitted into the trial (LOC xTR)

Number of cases within and across cells for table A_2.

TR	HP	GP	Across group N
COMB	N = 16	N = 14	N = 30
CBT	N = 17	N = 13	N = 30
D	N = 16	N = 12	N = 28
Within group N	N = 49	N = 39	Total N = 88

Variable	TR	HP ($\bar{x}$, SD)	GP ($\bar{x}$, SD)	Across group ($\bar{x}$, SD)	Analysis of variance Significance levels
AGE	COMB	47.4 ± 9.7	38.9 ± 8.2	43.4 ± 9.8	
	CBT	40.5 ± 10.4	37.6 ± 12.5	39.2 ± 11.2	LOC NS
	D	43.6 ± 12.9	42.9 ± 7.4	43.3 ± 10.7	TR NS
					LOC x TR NS
	Within group	43.7 ± 11.2	39.7 ± 9.6	42 ± 10.7	
* EDUC	COMB	2.2 ± 1.2	1.6 ± 0.9	1.9 ± 1.1	
	CBT	2.6 ± 1.2	1.2 ± 0.6	2.0 ± 1.2	LOC p = <.001
	D	2.1 ± 0.95	1.3 ± 0.6	1.8 ± 0.9	TR NS
					LOC x TR NS
	Within group	2.3 ± 1.1	1.4 ± 0.7	1.9 ± 1.1	
* SOC	COMB	2.6 ± 0.8	3.4 ± 1.3	2.9 ± 1.1	
	CBT	2.4 ± 1.1	3.5 ± 0.9	2.7 ± 1.1	LOC p = < .001
	D	2.6 ± 0.9	3.8 ± 0.93	3.1 ± 1.1	TR NS
					LOC x TR NS
	Within group	2.5 ± 0.91	3.6 ± 1.1	3.0 ± 1.1	

Variable		HP ($\bar{x}$, SD)	GP ($\bar{x}$, SD)	Across group (x, SD)	Analysis of variance Significance levels
* DILL	COMB	127.7 ± 117.2	22.3 ± 22.1	78.5 ± 100.9	
	CBT	112.9 ± 113.9	48.2 ± 47.84	84.7 ± 95.8	LOC $p < .001$
	D	32.4 ± 40.9	41.8 ± 37.2	36.4 ± 38.9	TR $p < .04$
	Within group	91.4 ± 104.9	36.9 ± 37.7	67.3 ± 85.9	LOC × TR $p < .02$
* PSE	COMB	34.1 ± 7.4	28.4 ± 5.8	31.5 ± 7.2	
	CBT	36.3 ± 8.0	28.0 ± 5.7	32.9 ± 8.2	LOC $p < .002$
	D	34.7 ± 11.7	31.6 ± 5.6	33.4 ± 9.5	TR NS
	Within group	35.1 ± 9.1	29.3 ± 5.8	32.5 ± 8.2	LOC × TR NS
BDI	COMB	29.0 ± 7.5	22.9 ± 4.9	26.3 ± 7.1	
	CBT	23.6 ± 7.1	25.7 ± 7.1	24.6 ± 7.2	LOC NS
	D	26.7 ± 7.7	28.6 ± 7.3	27.5 ± 7.7	TR NS
	Within group	26.4 ± 7.6	25.7 ± 6.9	26.1 ± 7.8	LOC × TR NS
HS	COMB	14.2 ± 5.2	10.6 ± 5.4	12.6 ± 5.5	
	CBT	13.6 ± 5.5	12.4 ± 5.4	13.1 ± 5.4	LOC NS
	D	13.5 ± 4.3	12.4 ± 4.1	13.1 ± 4.2	TR NS
	Within group	13.8 ± 4.9	11.8 ± 4.9	12.9 ± 5.0	LOC × TR NS
DEP	COMB	9.2 ± 2.8	9.6 ± 3.1	9.3 ± 2.9	
	CBT	8.2 ± 2.9	9.6 ± 2.5	8.8 ± 2.8	LOC NS
	D	9.2 ± 3.0	8.9 ± 2.4	9.1 ± 2.7	TR NS
	Within group	8.6 ± 2.9	9.4 ± 2.6	9.1 ± 2.8	LOC × TR NS
ANX	COMB	10.4 ± 2.3	9.5 ± 2.5	10.0 ± 2.4	
	CBT	9.5 ± 2.7	9.8 ± 3.0	9.6 ± 2.8	LOC NS
	D	10.0 ± 2.9	9.9 ± 1.5	10.0 ± 2.9	TR NS
	Within group	10.0 ± 2.9	9.7 ± 2.4	9.9 ± 2.7	LOC × TR NS
IN	COMB	7.2 ± 3.4	6.5 ± 3.5	6.9 ± 3.3	
	CBT	7.4 ± 3.5	7.0 ± 2.2	7.2 ± 3.0	LOC NS
	D	7.1 ± 2.9	7.3 ± 2.8	7.1 ± 2.9	TR NS
	Within group	7.2 ± 3.2	6.9 ± 2.8	7.2 ± 3.0	LOC × TR NS
OUT	COMB	6.2 ± 3.9	6.0 ± 2.9	6.1 ± 3.5	
	CBT	6.3 ± 3.2	6.7 ± 2.5	6.5 ± 2.9	LOC NS
	D	5.8 ± 3.4	9.6 ± 2.5	7.4 ± 3.6	TR NS
	Within group	6.1 ± 3.5	7.4 ± 3.0	6.6 ± 3.3	LOC × TR NS
IRR	COMB	13.3 ± 6	12.5 ± 5	12.9 ± 5.5	
	CBT	13.6 ± 6	13.7 ± 3.6	13.7 ± 5	LOC NS
	D	12.9 ± 5.9	16.9 ± 3.5	14.5 ± 5.4	TR NS
	Within group	13.3 ± 5.8	14.3 ± 4.4	13.7 ± 5.3	LOC × TR NS

Variable		HP ($\bar{x}$, SD)	GP ($\bar{x}$, SD	Across group ($\bar{x}$, SD)	Analysis of variance Significance levels
* AM	COMB	28.8 ± 12.9	39.0 ± 12.4	33.1 ± 13.5	
	CBT	34.2 ± 11.7	39.8 ± 10.9	36.7 ± 11.5	LOC $p < .03$
	D	32.3 ± 14.9	35.3 ± 11.1	33.5 ± 13.3	TR NS
					LOC × TR NS
	Within group	31.8 ± 13.1	38.2 ± 11.3	34.5 ± 12.7	
WAS	COMB	70.6 ± 12.2	64.6 ± 15.3	68.1 ± 13.7	
	CBT	63.7 ± 7.7	68.7 ± 13.2	65.9 ± 10.6	LOC NS
	D	62.4 ± 17.3	66.9 ± 11.0	64.3 ± 15.0	TR NS
					LOC × TR NS
	Within group	65.6 ± 13.1	66.8 ± 13.1	66.1 ± 13.1	
WD	COMB	82.3 ± 28	78.7 ± 6.8	80.8 ± 5.1	
	CBT	77.6 ± 5.5	79.9 ± 3.6	78.6 ± 4.9	LOC NS
	D	75.7 ± 13.9	80.2 ± 4.3	77.6 ± 11.2	TR NS
					LOC × TR NS
	Within group	78.5 ± 8.9	79.7 ± 5.0	79.0 ± 7.5	
ENV	COMB	32.8 ± 17.2	37.1 ± 14.5	34.7 ± 16	
	CBT	42.1 ± 17.9	38.9 ± 14.8	40.9 ± 16.4	LOC NS
	D	40.1 ± 18.5	34.0 ± 20.9	37.8 ± 19.4	TR NS
					LOC × TR NS
	Within group	38.5 ± 19.0	36.7 ± 16.5	37.8 ± 17.3	
FUT	COMB	31.8 ± 20.5	46.4 ± 20.1	38.0 ± 21.3	
	CBT	40.2 ± 20	44.6 ± 23.4	42.2 ± 21.5	LOC NS
	D	46.8 ± 19.2	41.1 ± 23.0	44.5 ± 20.6	TR NS
					LOC × TR NS
	Within group	39.6 ± 20.4	44.2 ± 21.7	41.5 ± 21.0	
IDS	COMB	53.2 ± 13.0	39.8 ± 12.6	47.4 ± 14.3	
	CBT	42.7 ± 12.6	39.8 ± 12.6	41.9 ± 12.5	LOC NS
	D	43.4 ± 17.3	45.1 ± 11.5	44.1 ± 15	TR NS
					LOC × TR NS
	Within group	46.3 ± 14.9	41.4 ± 12.2	44.2 ± 13.9	
IDW	COMB	11.6 ± 11.1	14.1 ± 10.9	12.7 ± 10.8	
	CBT	13.4 ± 7.6	11.2 ± 11.9	12.4 ± 9.6	LOC NS
	D	15.1 ± 14.8	13.4 ± 10.5	14.4 ± 13.0	TR NS
					LOC × TR NS
	Within group	13.5 ± 11.3	12.9 ± 10.9	13.2 ± 11.1	
WASE	COMB	41.9 ± 14.6	25.7 ± 16.6	34.9 ± 17.2	
	CBT	29.5 ± 11.1	28.8 ± 17.6	29.2 ± 14.0	LOC NS
	D	31.4 ± 15.7	31.6 ± 12.1	31.5 ± 14.1	TR NS
					LOC × TR NS
	Within group	34.1 ± 14.7	28.6 ± 15.5	32.0 ± 15.1	

Variable	TR	HP ($\bar{x}$, SD)	GP ($\bar{x}$, SD)	Across group (x, SD)	Analysis of variance Significance levels
* HRS	COMB	19.9 ± 4.5	21.2 ± 4.0	20.5 ± 4.3	
	CBT	16.8 ± 4.7	17.7 ± 4.0	17.2 ± 4.3	LOC NS
	D	17.3 ± 4.9	18.8 ± 3.7	17.9 ± 4.4	TR $p < .02$
	Within group	17.9 ± 4.8	19.3 ± 4.1	18.5 ± 4.5	LOC x TR NS
CS	COMB	6.2 ± 1.8	6.1 ± 2.0	6.1 ± 1.9	
	CBT	5.9 ± 3.6	6.2 ± 1.8	6.1 ± 2.9	LOC NS
	D	5.4 ± 2.3	6.9 ± 2.3	6.0 ± 2.3	TR NS
	Within group	5.8 ± 2.7	6.3 ± 2.0	6.1 ± 2.4	LOC x TR NS
WS	COMB	45.1 ± 14.0	51.1 ± 18.7	47.6 ± 16.2	
	CBT	56.7 ± 13.8	47.6 ± 14.6	52.8 ± 14.7	LOC NS
	D	40.2 ± 18.1	41.7 ± 12.5	40.8 ± 15.9	TR $p < .03$
	Within group	47.5 ± 16.7	46.8 ± 15.6	47.3 ± 16.2	LOC x TR NS

* Variables which significantly differentiated the groups at $p < .05$ or less

3. Comparison of completers and noncompleters

The next stage of the analysis was concerned with the investigation of possible differences between subgroups of patients who completed the trial and those who for various reasons dropped out of treatment. This was accomplished in two ways. A 2 x 2 x^2 analysis (two-tailed test) was calculated for total number of drop-outs from treatment in each location, the point being to examine the possible relationship between attrition and the different clinics. Next, a two-way anslysis of variance was computed for all clinical, demographic, and baseline measures of severity of depression.

Table $6A_3$ shows that, while attrition was considerably higher in the general practice sample, the relationship between location and attrition was not statistically significant.

The nine hospital and 15 general practice drop-outs were equally

distributed among the three treatments, i.e. 3, 3 and 3, and 5, 5 and 5. Thus, no one treatment can be said to have been more liable to attrition.

Table $6A_3$ X^2 analysis for completers and noncompleters

	Completers	Noncompleters	Totals
H.P.	40	9	49
G.P.	24	15	39
Totals	64	24	88

$x^2 = 3.47$, df = 1, NS

The results of the analysis of variance for the subgroups of completers and noncompleters are presented in Table $6A_4$ (for additional information see Appendix XIII).

There were 22 completers in each of the cognitive therapy groups and 20 completers in the pharmacotherapy group, making a total of 64 completers in the trial.

Six variables significantly differentiated the patients who completed treatment from those who did not. Of these, five variables were measures of severity of illness. Noncompleters had higher mean scores on both self-reported (BDI) and observer-rated (HRS) depression, were more inwardly irritable on average, had higher mean total irritability scores, and were more negative in terms of their mean score on self-reported view of the environment. Those who did not complete treatment were also less educated than patients who had a full course of treatment.

Since this analysis was computed on the same population as before,

the treatment differences (DILL, HRS, WS) are identical to those reported in the previous section.

The results also indicate that there were no status (i.e. completers, noncompleters) by treatment interactions.

Table $6A_4$ Results of analysis of variance on completers and non-completers (Status x TR)

Number of cases within and across cells for table $6A_4$

TR	Completers	Noncompleters	Across group N
COMB	N = 22	N = 8	N = 30
CBT	N = 22	N = 8	N = 30
D	N = 20	N = 8	N = 28
Within group N	64	24	Total N = 88

Variable	TR	Completers $\bar{x}$, SD	Noncompleters $\bar{x}$, SD	Across group $\bar{x}$, SD	Analysis of variance Significance levels
AGE	COMB	45.4 ± 9.8	38.0 ± 8.3	43.4 ± 9.8	
	CBT	39.7 ± 11.1	37.8 ± 12.2	39.2 ± 11.2	Status NS
	D	44.9 ± 10.8	39.4 ± 10.1	43.3 ± 10.7	TR NS
	Within group	43.3 ± 10.7	38.4 ± 9.9	42 ± 10.7	Status x TR NS
* EDUC	COMB	1.9 ± 1.1	2.0 ± 1.1	1.9 ± 1.1	
	CBT	2.3 ± 1.2	1.4 ± 0.74	2.0 ± 1.2	Status p <.05
	D	2.0 ± 0.9	1.3 ± 0.5	1.8 ± 0.9	TR NS
	Within group	2.0 ± 1.1	1.5 ± 0.83	1.9 ± 1.1	Status x TR NS
SOC	COMB	3.0 ± 1.2	2.8 ± 1.0	2.9 ± 1.1	
	CBT	2.7 ± 1.2	3.4 ± 0.5	2.7 ± 1.1	Status NS
	D	3.0 ± 0.9	3.5 ± 1.3	3.1 ± 1.1	TR NS
	Within group	2.8 ± 1.1	3.2 ± 1.0	3.0 ± 1.1	Status x TR NS
* DILL	COMB	75.5 ± 75.5	86.9 ± 158.1	78.5 ± 100.9	
	CBT	89.5 ± 105.3	72.0 ± 66.9	84.7 ± 95.8	Status NS
	D	38.5 ± 41.3	31.5 ± 34.3	36.4 ± 38.9	TR p < .04
	Within group	68.7 ± 81.1	63.5 ± 99.5	67.3 ± 85.9	Status x TR NS

Variable	TR	Completers $\bar{x}$, SD	Noncompleters $\bar{x}$, SD	Across group $\bar{x}$, SD	Analysis of Variance Significance levels
PSE	COMB	32.3 ± 7.9	29.3 ± 5.1	31.5 ± 7.2	
	CBT	32.0 ± 6.9	34.6 ± 11.3	32.7 ± 8.2	Status NS
	D	32.9 ± 10.6	34.9 ± 6.7	33.4 ± 9.5	TR NS
	Within group	32.4 ± 8.4	32.9 ± 8.2	32.5 ± 8.3	Status x TR NS
* BDI	COMB	25.8 ± 7.3	27.7 ± 6.6	26.3 ± 7.1	
	CBT	21.9 ± 4.6	32.0 ± 8.2	24.6 ± 7.2	Status $p < .001$
	D	24.7 ± 6.6	34.5 ± 4.0	27.5 ± 7.4	TR NS
	Within group	24.1 ± 6.4	31.6 ± 6.8	26.1 ± 7.3	Status x TR NS
HS	COMB	12.0 ± 5.6	14.1 ± 5.3	12.6 ± 5.5	
	CBT	12.4 ± 5.5	14.8 ± 5.0	13.1 ± 5.4	Status NS
	D	12.8 ± 4.4	14.0 ± 3.6	13.1 ± 4.2	TR NS
	Within group	12.4 ± 5.2	14.3 ± 4.5	12.9 ± 5.0	Status x TR NS
DEP	COMB	9.4 ± 2.9	9.0 ± 3.2	9.3 ± 2.9	
	CBT	8.0 ± 2.6	11.0 ± 2.3	8.8 ± 2.8	Status NS
	D	8.9 ± 3.0	9.7 ± 1.7	9.1 ± 2.7	TR NS
	Within group	8.8 ± 2.8	10.0 ± 2.4	9.1 ± 2.8	Status x TR NS
ANX	COMB	10.0 ± 2.6	10.2 ± 1.8	10.0 ± 2.4	
	CBT	9.2 ± 2.8	10.9 ± 2.7	9.6 ± 2.8	Status NS
	D	9.7 ± 3.2	10.9 ± 1.8	10.0 ± 2.9	TR NS
	Within group	9.6 ± 2.8	10.7 ± 2.1	9.9 ± 2.7	Status x TR NS
* IN	COMB	6.8 ± 3.5	7.2 ± 3.1	6.9 ± 3.3	
	CBT	6.5 ± 2.9	9.0 ± 2.1	7.2 ± 3.0	Status $p < .03$
	D	6.6 ± 2.7	8.7 ± 2.8	9.1 ± 2.9	TR NS
	Within group	6.7 ± 3.0	8.4 ± 2.6	7.1 ± 3.0	Status x TR NS
OUT	COMB	6.1 ± 3.4	5.8 ± 4.1	6.1 ± 3.5	
	CBT	5.9 ± 2.5	8.0 ± 3.5	6.5 ± 2.9	Status NS
	D	6.9 ± 3.6	8.6 ± 3.5	7.4 ± 3.6	TR NS
	Within group	6.3 ± 3.2	7.6 ± 3.6	6.6 ± 3.3	Status x TR NS
* IRR	COMB	13 ± 5.4	13 ± 6.3	12.2 ± 5.5	
	CBT	12.5 ± 4.7	17 ± 4.7	13.7 ± 5.0	Status $p < .03$
	D	13.6 ± 5.2	17 ± 5.1	14.5 ± 5.4	TR NS
	Within group	12.9 ± 5.1	15.9 ± 5.4	13.7 ± 5.3	Status x TR NS

Variable	TR	Completers $\bar{x}$, SD	Noncompleters $\bar{x}$, SD	Across group $\bar{x}$, SD	Analysis of variance Significance levels
AM	COMB	34.2 ± 13.3	29.3 ± 14.6	33.1 ± 13.5	
	CBT	38.6 ± 10.4	31.3 ± 13.4	36.9 ± 11.5	Status NS
	D	34.8 ± 13.7	29.7 ± 12.4	33.5 ± 13.3	TR NS
					Status x TR NS
	Within group	36.0 ± 12.5	30.2 ± 12.8	34.5 ± 12.7	
WAS	COMB	68.6 ± 13.1	66.0 ± 16.7	68.1 ± 13.7	
	CBT	66 ± 9.7	65.5 ± 13.4	65.9 ± 10.6	Status NS
	D	64.1 ± 15	64.9 ± 16.1	64.3 ± 15	TR NS
					Status x TR NS
	Within group	66.3 ± 12.7	65.4 ± 14.5	66.1 ± 13.1	
WD	COMB	81.1 ± 4.0	79.5 ± 8.6	80.8 ± 5.1	
	CBT	77.9 ± 5.1	80.8 ± 3.5	78.6 ± 4.9	Status NS
	D	78.5 ± 6.0	75.1 ± 20.4	77.6 ± 11.2	TR NS
					Status x TR NS
	Within group	79.2 ± 5.2	78.5 ± 12.4	79.0 ± 7.5	
* ENV	COMB	36.2 ± 17.5	29.3 ± 7.6	34.7 ± 16	
	CBT	44.2 ± 15.7	31.3 ± 15.4	40.7 ± 16.4	Status $p < .02$
	D	40.6 ± 16.2	29.6 ± 26.5	37.8 ± 19.4	TR NS
					Status x TR NS
	Within group	40.3 ± 16.5	30.1 ± 17.6	37.8 ± 17.3	
FUT	COMB	37.9 ± 21.2	38.5 ± 23.6	38 ± 21.3	
	CBT	46.3 ± 20.5	30.9 ± 20.3	42.2 ± 21.2	Status NS
	D	46.1 ± 19.2	40 ± 25.2	44.5 ± 20.6	TR NS
					Status x TR NS
	Within group	43.3 ± 20.4	36.1 ± 22.2	41.5 ± 21.0	
IDS	COMB	46.7 ± 14.2	50.2 ± 15.9	47.4 ± 14.3	
	CBT	38.7 ± 10.9	49 ± 14.1	41.4 ± 12.5	Status NS
	D	43.6 ± 14.7	45.4 ± 16.7	44.1 ± 15	TR NS
					Status x TR NS
	Within group	42.9 ± 13.5	48.1 ± 14.9	44.2 ± 13.9	
IDW	COMB	12.5 ± 11.4	13.5 ± 9.5	12.7 ± 10.8	
	CBT	11.8 ± 9.0	14.0 ± 11.5	12.4 ± 9.6	Status NS
	D	14.4 ± 13.3	14.6 ± 13.2	14.4 ± 13.0	TR NS
					Status x TR NS
	Within group	12.9 ± 11.2	14.0 ± 11.0	13.2 ± 11.1	
WASE	COMB	34.5 ± 17.5	36.7 ± 17.9	34.9 ± 17.2	
	CBT	27.4 ± 13.5	34.3 ± 15.2	29.2 ± 14	Status NS
	D	30.2 ± 14.8	35.1 ± 12	31.5 ± 14.1	TR NS
					Status x TR NS
	Within group	30.7 ± 15.4	35.2 ± 14.3	32 ± 15.1	

Variable		Completers $\bar{x}$, SD	Noncompleters $\bar{x}$, SD	Across group $\bar{x}$, SD	Analysis of variance Significance levels
* HRS	COMB	20.5 ± 4.6	20.4 ± 3.7	20.5 ± 4.3	
	CBT	15.9 ± 3.5	20.8 ± 4.5	17.2 ± 4.3	Status $p < .005$
	D	17.4 ± 4.6	19.1 ± 3.9	17.9 ± 4.7	TR $p < .02$
	Within group	18 ± 4.5	20.1 ± 3.9	18.5 ± 4.5	Status x TR NS
CS	COMB	6.0 ± 1.9	6.9 ± 2.0	6.1 ± 1.9	
	CBT	6.2 ± 3.2	5.8 ± 1.9	6.1 ± 2.9	Status NS
	D	5.9 ± 2.3	6.3 ± 2.7	6.0 ± 2.3	TR NS
	Within group	6.0 ± 2.5	6.3 ± 2.1	6.1 ± 2.4	Status x TR NS
* WS	COMB	45.4 ± 15.1	56 ± 18.6	47.6 ± 16.2	
	CBT	53.9 ± 15	49.6 ± 14	52.8 ± 14.7	Status NS
	D	40.9 ± 16.6	41 ± 14.5	40.8 ± 15.9	TR $p < .03$
	Within group	46.9 ± 16.3	48.4 ± 16	47.3 ± 16.2	Status x TR NS

* Variables which significantly differentiated the groups at $p < .05$ or less

4. Comparison of completers only in the hospital and general practice samples

Having examined the characteristics of the entire sample in a number of ways and the ways in which completers and drop-outs differed, the characteristics of those patients who completed the trial were analysed. The reasons for this were: 1) to make the characteristics of this final sample more easily comparable with the characteristics of the entire population admitted to the trial, and 2) to ascertain whether any of these factors were of sufficient importance to demand further consideration when analysing outcome.

The section opens with a summary of the population studied and a baseline description of completers only which includes only selected variables. Next, the results of two-way analysis of variance

(location x treatment) for all variables are reported. This is followed by a breakdown of the populations in terms of length of time in therapy and number of therapy sessions.

Table $6A_5$ summarises the population studied. To recapitulate, 140 patients were assessed, of whom 52 were rejected. There was a somewhat higher rate of rejection in the general practice but this was not significant. Also, attrition was higher in the general practice group. A total of 40 hospital patients completed the trial compared to 24 completers in general practice.

Table $6A_5$ Population studied

	Hospital O.P.	General Practice	
Nos. assessed	71	69	140
* Nos. rejected	22 (31%)	30 (43%)	52
Drop-outs	9 (18%)	15 (38%)	24
Completers	40	24	64

* $x^2 = 1.83$, N.S. Df = 1

A baseline description for completers only is presented in table $6A_6$. The table includes some data from the results of the analysis of variance. There was no difference in age distribution, but socio-economic level and education differed at a high significance level, the hospital patients being of a higher socio-economic level (Registrar General Classification) and of a higher educational level (coded as 1 = left school at minimal age; 2 = at least 2 O levels or completed secondary education; 3 = special training such as technical education; 4 = at least some university education). The hospital patients had suffered more depressive episodes and their current episode was of longer

duration.

There were no significant differences in basal BDI and HRS-D ratings, but the hospital patients had a significantly higher total severity of symptoms on the PSE.

Table $6A_6$ Baseline description for completers only

	H.P. (N = 40)			G.P. (N = 24)		
	M	± S.D.	(Range)	M	± S.D.	(range)
Age	44.5	± 11.4	(21-63)	41.3	± 9.5	(24-57)
Socio-economic *** level	2.4	± 0.9	(1-4)	3.7	± 1	(1-5)
Education ***	2.5	± 1.1	(1-4)	1.3	± 0.6	(1-3)
Previous dep. ** episodes	3.1	± 4.43	(0-25)	0.8	± 0.96	(0-3)
Duration of illness * (weeks)	86.8	± 93.7	(4-416)	38.7	± 39.9	(3-156)
BDI	24.6	± 7	(15-40)	23.3	± 5.3	(14-34)
HRS-D	17.3	± 4.6	(11-33)	19.0	± 4.5	(11-26)
PSE total *	34.1	± 8.9	(18-51)	29.5	± 6.6	(20-46)

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Table $6A_7$ shows the results of the analysis of variance (LOC x TR) for baseline differences for all variables, some of which were presented in the previous table (for further details see Appendix XIV). Apart from the differences discussed earlier, it was found that hospital patients were, on the whole, more hopeless, less outwardly irritable and had lower self esteem than general practice patients. There was only one treatment difference: the combination group had a higher mean HRS-D score than the drug group who in turn were higher than the CBT group.

The hospital patients therefore are a sicker population than the general practice patients. They are more chronically ill, suffer more recurrent episodes, have more severe symptoms, are more hopeless and self-critical. They are, however, less outwardly irritable than the general practice patients.

Table $6A_7$ Results of analysis of variance on completers only (LOC x TR)

Number of cases within and across cells for table $6A_7$

TR	HP	GP	Across group N
COMB	N = 13	N = 9	N = 22
CBT	N = 14	N = 8	N = 22
D	N = 13	N = 7	N = 20
Within group N	40	24	64

Variable	TR	HP $\bar{x}$, SD	GP $\bar{x}$, SD	Across group x, SD	Analysis of Variance Significance Levels
AGE	COMB	49.2 ± 8.8	39.9 ± 8.7	45.4 ± 9.8	
	CBT	39.3 ± 10.5	40.4 ± 12.8	39.7 ± 11.1	LOC NS
	D	45.4 ± 12.9	44.0 ± 6.1	44.9 ± 10.8	TR NS
	Within group	44.5 ± 11.4	41.3 ± 9.5	43.3 ± 10.6	LOC x TR NS
* EDUC	COMB	2.3 ± 1.3	1.2 ± 0.44	1.9 ± 1.1	
	CBT	2.9 ± 1.1	1.3 ± 0.70	2.3 ± 1.2	LOC $p < .001$
	D	2.3 ± .94	1.4 ± 0.78	2.0 ± 0.97	TR NS
	Within group	2.5 ± 1.1	1.3 ± 0.62	2.0 ± 1.2	LOC x TR NS
* SOC	COMB	2.5 ± 0.87	3.7 ± 1.2	3.0 ± 1.2	
	CBT	2.1 ± 1.0	3.6 ± 1.1	2.7 ± 1.2	LOC $p < .001$
	D	2.6 ± 0.76	3.7 ± 0.95	3.0 ± 0.97	TR NS
	Within group	2.4 ± 0.90	3.7 ± 1.0	2.9 ± 1.1	LOC x TR NS
* DILL	COMB	110.4 ± 79.8	25 ± 23.3	75.5 ± 75.5	
	CBT	111 ± 122.5	51.9 ± 53.7	89.5 ± 105.3	LOC, $p < .01$
	D	37 ± 44.2	41.4 ± 38.6	31.4 ± 41.3	TR NS
	Within group	86.8 ± 93.7	38.7 ± 39.9	68.7 ± 89	LOC x TR NS

Variable		HP $\bar{x}$, SD	GP $\bar{x}$, SD	Across group $\bar{x}$, SD	Analysis of variance Significance Levels
* PSE	COMB	34.5 ± 7.9	29.0 ± 6.6	32.3 ± 7.9	
	CBT	34.5 ± 6.2	27.6 ± 6.0	32.0 ± 6.9	LOC $p < .05$
	D	33.1 ± 12.3	32.4 ± 7.2	32.9 ± 10.6	TR NS
	Within group	34.1 ± 8.9	29.5 ± 6.6	32.4 ± 8.3	LOC x TR NS
BDI	COMB	28.0 ± 7.9	22.7 ± 5.1	25.8 ± 7.3	
	CBT	21.3 ± 4.9	22.8 ± 4.1	21.9 ± 4.6	LOC NS
	D	24.7 ± 6.7	24.7 ± 7.1	24.8 ± 6.6	TR NS
	Within group	24.6 ± 6.9	23.3 ± 5.3	24.1 ± 6.4	LOC x TR NS
* HS	COMB	13.9 ± 5.5	10.1 ± 5.4	12.0 ± 5.6	
	CBT	13.5 ± 5.5	10.6 ± 5.4	12.4 ± 5.5	LOC $p < .05$
	D	13.4 ± 4.6	11.6 ± 4.2	12.7 ± 4.4	TR NS
	Within group	13.4 ± 5.1	10.7 ± 4.3	12.4 ± 5.2	LOC x TR NS
DEP	COMB	9.3 ± 2.9	9.6 ± 3.0	9.4 ± 2.9	
	CBT	7.5 ± 2.7	9.0 ± 2.2	8.0 ± 2.6	LOC NS
	D	9.0 ± 3.2	8.7 ± 2.9	8.9 ± 3.0	TR NS
	Within group	8.6 ± 3	9.1 ± 2.6	8.8 ± 2.8	LOC x TR NS
ANX	COMB	10.3 ± 2.4	9.6 ± 2.9	10 ± 2.6	
	CBT	9.1 ± 2.8	9.4 ± 2.9	9.2 ± 2.8	LOC NS
	D	9.6 ± 2.8	9.8 ± 1.5	9.7 ± 3.2	TR NS
	Within group	9.6 ± 3	9.5 ± 2.5	9.6 ± 2.8	LOC x TR NS
IN	COMB	6.9 ± 3.7	6.7 ± 3.4	6.8 ± 3.5	
	CBT	6.6 ± 3.4	6.4 ± 2.3	6.5 ± 2.9	LOC NS
	D	6.5 ± 2.9	6.9 ± 2.5	6.6 ± 2.7	TR NS
	Within group	6.7 ± 3.3	6.6 ± 2.7	6.6 ± 3	LOC x TR NS
* OUT	COMB	5.6 ± 3.8	6.8 ± 2.7	6.1 ± 3.4	
	CBT	5.4 ± 2.7	6.8 ± 1.9	5.9 ± 2.5	LOC $p < .008$
	D	5.5 ± 3.2	9.6 ± 3.0	6.9 ± 3.6	TR NS
	Within group	5.5 ± 3.2	7.6 ± 2.8	6.3 ± 3.2	LOC x TR NS
IRR	COMB	12.5 ± 6.1	13.5 ± 4.6	12.9 ± 5.5	
	CBT	12 ± 5.3	13.1 ± 3.4	12.5 ± 4.7	LOC NS
	D	12 ± 5.6	16.4 ± 3.0	13.5 ± 5.2	TR NS
	Within group	12.2 ± 5.6	14.3 ± 3.9	13.0 ± 5.1	LOC x TR NS

Variable		HP $\bar{x}$, SD	GP $\bar{x}$, SD	Across group $\bar{x}$, SD	Analysis of Variance Significance levels
	COMB	30.2 ± 13.8	39.8 ± 10.9	34.2 ± 13.3	
*	CBT	35.5 ± 10.8	44.1 ± 7.2	38.6 ± 10.4	LOC p < .05
AM	D	34.8 ± 15.2	35 ± 11.2	34.9 ± 13.7	TR NS
					LOC x TR NS
	Within group	33.6 ± 13.2	39.9 ± 10.2	35.9 ± 12.5	
	COMB	70.2 ± 12.9	66.3 ± 13.9	68.6 ± 13.1	
	CBT	64.1 ± 7.3	69.4 ± 12.7	66 ± 9.7	LOC NS
WAS	D	61.7 ± 17.4	68.3 ± 8.6	64.1 ± 15	TR NS
					LOC x TR NS
	Within group	65.3 ± 13.3	67.9 ± 11.7	66.3 ± 12.7	
	COMB	82.1 ± 3.1	79.7 ± 4.8	87.1 ± 4	
	CBT	77.1 ± 5.7	79.1 ± 3.9	77.9 ± 5.1	LOC NS
WD	D	78.1 ± 6.7	79.1 ± 4.9	78.5 ± 6	TR NS
					LOC x TR NS
	Within group	79.1 ± 5.7	79.3 ± 4.4	79.2 ± 5.2	
	COMB	34.1 ± 18.9	39.2 ± 15.7	36.2 ± 17.5	
	CBT	45.6 ± 15.7	41.8 ± 16.6	44.2 ± 15.7	LOC NS
ENV	D	45.3 ± 16.3	31.8 ± 12.5	40.6 ± 16.2	TR NS
					LOC x TR NS
	Within group	41.78 ± 17.4	37.9 ± 15	40.3 ± 16.5	
	COMB	31.9 ± 21.9	46.5 ± 17.7	37.9 ± 21.2	
	CBT	42.9 ± 18.1	52.3 ± 24.1	46.3 ± 20.5	LOC NS
FUT	D	45.5 ± 19.7	47 ± 19.9	46.1 ± 19.2	TR NS
					LOC x TR NS
	Within group	40.2 ± 20.3	49 ± 19.9	43.3 ± 20.4	
	COMB	51.5 ± 13.8	39.8 ± 12.2	46.7 ± 14.2	
	CBT	40.8 ± 11.5	35 ± 9.3	38.7 ± 10.9	LOC NS
IDS	D	43.3 ± 16.6	44 ± 11.6	43.6 ± 14.8	TR NS
					LOC x TR NS
	Within group	45.1 ± 14.5	39.5 ± 11.3	43 ± 13.5	
	COMB	11.8 ± 11.7	13.3 ± 11.5	12.5 ± 11.9	
	CBT	13.1 ± 7.8	9.7 ± 11.2	11.9 ± 9.0	LOC NS
IDW	D	16.3 ± 15.9	10.9 ± 5.6	14.4 ± 13.3	TR NS
					LOC x TR NS
	Within group	13.7 ± 12	11.4 ± 9.7	12.9 ± 11.2	
	COMB	40 ± 15.6	26.4 ± 17.7	34.5 ± 17.5	
	CBT	28.6 ± 11.8	25.3 ± 16.7	27.4 ± 13.5	LOC NS
WASE	D	28.5 ± 14.9	33.3 ± 15.2	30.2 ± 14.8	TR NS
					LOC x TR NS
	Within group	32.3 ± 14.8	28 ± 16.3	30.7 ± 15.4	

Variable	TR	HP $\bar{x}$, SD	GP $\bar{x}$, SD	Across group $\bar{x}$, SD	Analysis of Variance Significance Level
	COMB	20 ± 4.9	21.2 ± 4.0	20.5 ± 4.6	
*	CBT	15.7 ± 3.5	16.3 ± 3.8	15.9 ± 3.5	LOC NS
HRS	D	16.4 ± 4.3	19.3 ± 4.8	17.4 ± 4.6	TR $p < .004$
	Within group	17.3 ± 4.6	19 ± 4.5	17.9 ± 4.6	LOC x TR NS
	COMB	5.9 ± 1.9	5.9 ± 1.9	5.9 ± 1.9	
	CBT	6.1 ± 3.8	6.2 ± 1.9	6.0 ± 3.1	LOC NS
CS	D	5.5 ± 3.3	6.5 ± 2.2	5.4 ± 2.3	TR NS
	Within group	5.9 ± 2.8	6.2 ± 1.9	6.1 ± 2.5	LOC x TR NS
	COMB	45.0 ± 14.4	45.6 ± 17	45.4 ± 15	
	CBT	56.2 ± 14.3	50 ± 16	54 ± 15	LOC NS
WS	D	40.3 ± 19.4	42 ± 11	40 ± 16.6	TR NS
	Within group	47.5 ± 17.2	46 ± 19.9	46.9 ± 16.3	LOC x TR NS

* Variables which significantly differentiated the group at $p = .05$ or less

From table $6A_8$, 22 patients had combination treatment (13 HP and 9 GP), 22 had CBT (14 HP and 8 GP), 20 had drugs (13 HP and 7 GP).

Table $6A_8$ Breakdown of populations according to treatment

	N	HP (N = 40) weeks in trial	Sessions	N	GP (N = 24) weeks in trial	Sessions	N
COMB	13	15.2 ± 4.3	16.6 ± 5.1	9	11.6 ± 4.1	11.6 ± 4.1	22
CBT	14	12.4 ± 4.8	16.9 ± 7.3	8	12.4 ± 4.6	12.5 ± 5	22
D	13	12.3 ± 4.0	-	7	12.7 ± 2.6	-	20

Weeks: HP/COMB > GP/COMB, t = 1.91, Df 20, NS

Sessions: HP/COMB > GP/COMB, t = 2.54, Df 20, $p < .05$

HP/CBT > GP/CBT, t = 1.67, Df 20, NS

The HP combination group was in treatment for slightly longer but the difference in weeks from the other groups is not significant.

They, however, had significantly more treatment sessions than the GP combinative group. The HP/CBT group also had more treatment sessions than the GP/CBT group, but the difference is not statistically significant. Thus, the hospital patients were given more treatment sessions than the general practice patients.

5. Correlation of baseline measures

This section is directed towards examining the interrelationships between demographic, clinical and severity measures of depression which have been selected for the present study. The main interest, however, was to show that the dependent measures have concurrent validity. Concurrent validity is the demonstration that independent techniques of assessment measure the same phenomena.

Pearson product moment correlations were computed to assess the degree of association between the various measures of mood, cognition and psychomotor inefficiency. Only significant baseline correlations are reported and these are based on data obtained from all patients admitted into the trial, i.e. completers and noncompleters from the two clinics.

Table $6A_9$ shows the significant correlations computed on baseline data between age, education, social class, total severity score on the PSE and the various measures of severity of depression. All of these significant correlations were low. The highest correlation was between level of education and social class (-.64). The negative sign reflects the rating system used in the study, i.e. the higher the numerical rating for education, the more educated a patient was, whereas the higher the rating for social class, the 'lower down' the patient was in terms of social standing.

Table $6A_9$ Significant baseline correlations between demographic factors, total score on the PSE and dependent measures for all patients admitted into the trial

N = 88

AGE		EDUC		SOC		PSE	
OUT	-.22 *	SOC	-.64 ***	OUT	.28 **	BDI	.47 ***
IRR	-.23 *	DILL	.31 **	IRR	.22 *	IN	.36 ***
		BDI	.23 *	WD	.29 **	OUT	.24 *
		OUT	-.39 ***	ENV	-.27 **	IRR	.36 ***
		IRR	-.36 ***	WS	-.30 **	AM	-.39 **
		HRS	-.31 **			IDS	.29 ***
		WS	.48 ***			WASE	.35 ***
						HRS	.39 ***

Df = 86 * p = < .05, ** p = < .01, *** p = < .001

Correlations between mood scales are presented in table $6A_{10}$.

Table $6A_{10}$ Significant baseline correlations between mood measures for all patients admitted into the trial

N = 88

BDI		HRS		DEP		ANX		IN		OUT	
HRS	.50 ***	DEP	.43 ***	ANX	.37 ***	IN	.40 ***	OUT	.38 ***	IRR	.84 ***
DEP	.30 **	ANX	.27 **	IN	.29 **	OUT	.27 **	IRR	.81 ***		
ANX	.39 ***	IN	.35 ***	IRR	.28 **	IRR	.40 ***				
IN	.40 ***	IRR	.25 *								
OUT	.24 *										
IRR	.38 ***										

Df = 86 * p = < .05, ** p = < .01, *** p = < .001

Again, nearly all of the correlations were low. However, they were significant and, with respect to the main measures (i.e. BDI and HRS), the correlation (.50) was of the order expected from, for example, the information reported by Carroll et al. (1973) and Prusoff et al. (1972) for studies with comparable self-report and observer scales.

These suggest that the degree of concordance between such measures is lower during the acute episode than at recovery. It is interesting that the depression subscale (DEP) of the IDA which consists of biological and affective items, correlates almost as highly with the HRS as the BDI and at the same high level of significance.

From table $6A_{11}$ showing the intercorrelations of the cognitive measures, hopelessness (HS) was correlated negatively at a high significance level with the semantic differential measures of each component in Beck's cognitive triad (AM, ENV, FUT). That is, the more hopeless a patient was according to his responses on the HS (high score), the more negatively he viewed himself, his environment and his personal future in terms of the concepts used in the semantic differential (low scores). While a stronger degree of association would have been desirable, the relatively high negative correlation between the two measures of hopelessness (HS and FUT, -.77) provides tentative support for the concurrent validity of the HS. This is important because the original version of Beck's HS has not been validated in a British population. In addition, there is a highly significant, positive interrelationship between the different components in the negative cognitive triad (AM and ENV, .60; AM and FUT .58; ENV and FUT .51).

The association between mood and cognitive measures for all patients admitted into the trial is presented in table $6A_{12}$. The BDI was consistently correlated at a high significance level with those cognitive measures which might be expected to reflect depressed thinking, including the degree of discrepancy between perceived current

Table $6A_{11}$ Significant baseline correlations between cognitive measures for all patients admitted into the trial

HS		AM		WAS		WD		ENV		FUT		IDS		IDW	
AM	-.62***	WAS	.24*	WD	.47***	IDS	.42**	FUT	.51***	IDS	-.57***	WASE	.70***	WASE	-.54***
ENV	-.53***	ENV	.60***	WASE	.61***	WASE	.27	IDS	-.54***	WASE	-.35***				
FUT	-.77***	FUT	.58***					WASE	-.48***						
IDS	.57***	IDS	-.84*												
WASE	.37	IDW	-.23***												
		WASE	-.60												

Df = 86 * p = < .05 ** p = < .01 *** p = < .001

Table $6A_{12}$ Significant baseline correlations between mood and cognitive measures for all patients admitted to the trial

(N = 88)

Mood Measures	Cognitive Measures					
	HS	AM	ENV	FUT	IDS	WASE
BDI	.47 ***	-.55 ***	-.48 ***	-.40 ***	-.51 ***	.45 ***
HRS	-	-.28 **	-	-	-	.35 ***
DEP	-	-.29 **	-.23 *	-	.30 **	.27 **
ANX	.28 ***	-.36 ***	-.33 **	-	.30 **	-
IN	.40 ***	-.41 ***	-	-.31 **	.38 ***	.29 **
OUT	-	-.24 *	-.23 *	-	.23 *	-
IRR	.32 **	-.39 ***	-.26 **	-.22 *	.36 ***	.22 *

Df = 86 * p = < .05 ** p = < .01 *** p = < .001

self and ideal self (IDS) and perceived deviation from previously 'well' self (WASE). However, the correlations were moderate, the highest being between BDI and AM (-.55) and the lowest between BDI and FUT (-.40). The association between depth of depression as assessed by BDI and hopelessness (HS) was not as strong (.47), for example, as that reported by Minkoff et al. (.68 (1973)). The lower correlation obtained in the present study might be attributed to the fact that none of the patients were psychotic or schizophrenic, and all were outpatients. In the Minkoff et al. study, the sample consisted of inpatients, a considerable proportion of whom had been severely disturbed.

With regard to the other self-report measures of mood, while the correlations were moderate to low, both anxiety (ANX) and inward directed irritability (IN) were correlated positively with hopelessness (HS) and negatively with low self esteem (AM). Moreover, total irritability score (IRR) was significantly correlated with cognitive aspects of depression.

The observer rating of depression (HRS) was associated with only two cognitive measures, self-esteem (AM) and perceived deviation from previous self (WASE). There were no significant relationships between HRS score and the other cognitive aspects of depression. This may reflect the fact that the HRS is loaded rather heavily with biological items and contains hardly any items which relate directly to the specific cognitive phenomena of interest here.

Finally, table $6A_{13}$ shows the significant basal correlations between the different behavioural measures and the rest of the variables.

Speech rate (CS) correlated significantly with the depression subscale of the IDA, but otherwise this measure, when compared across subjects, was not associated with the other aspects of depression measured in the study. On the other hand, writing speed (WS) was correlated negatively with four measures of mood (BDI, ANX, OUT, IRR) and two measures of cognition (AM, FUT). There was a highly significant, positive correlation between performance on the writing speed task and level of education (.48). The two behavioural measures, however, were not significantly correlated.

Table $6A_{13}$ Significant baseline correlations between behavioural, demographic and other dependent measures for all patients admitted into the trial

N = 88

CS			WS		
DEP	.28	**	EDUC	.48	***
			SOC	-.30	**
			BDI	-.23	*
			ANX	-.26	**
			OUT	-.33	**
			AM	.25	*
			FUT	-.22	*
			IRR	-.30	*

Df = 86 * p = < .05 ** p = < .01 *** p = < .001

These results indicate that, of the two behavioural measures, speech rate (CS) as assessed in this investigation, is the least sensitive measure of psychomotor inefficiency. While writing speed would appear to be slightly better, performance on this task is almost certainly related to how well educated a person is to begin with.

B. ANALYSIS OF OUTCOME

In keeping with the objectives of the research, it was decided to examine (1) whether each treatment group in each clinic actually improved from their initial mean scores in terms of the dependent variables (mood, cognitive and behavioural measures), and (2) to see how effective the different treatment modalities were relative to one another.

The main analysis used was analysis of variance (Winer, 1970). Specifically, a two-way analysis of covariance for change scores was computed, using as covariates the four descriptive variables which differentiated the two populations: i.e. duration of illness, education, socio-economic level and PSE total severity score. Change scores were obtained by simply subtracting the last score from the initial score.

Outcome was investigated in another way. End-point scores from the main self-report depression scale (BDI) were used to classify people into subgroups of responders and nonresponders, i.e. 'responders' obtained a BDI score ≤ 8 and/or a 50% reduction from initial score by the end of treatment. Then, Fisher's Exact Probability Test was employed to check the relationship between these categories and the two populations for each treatment modality.

Another major analysis was needed. In order to control for differences in scores at the time of admission into the trial, a two-way analysis of covariance for percentage change scores was computed, controlling for the same descriptive differences as in the analysis of change scores. Percentage change scores were computed by subtracting the last score from the initial score, then dividing

the difference by the initial score and multiplying by one-hundred.

It was hoped that these analyses would take into account any real differences between the samples from each clinic and among the three treatments.

Assessment of the significance of improvement in each treatment group in each clinic was computed by testing the significance of mean change and percentage change scores using a t test ($t = \frac{\text{mean change scores}}{(\frac{SD}{N})}$). Thus, for each of the dependent variables (18) a t value was obtained for each treatment group and the significance level checked to determine whether each treatment group had improved on average from their initial level of depression.

With regard to the relative effectiveness of the three treatments, a-posteriori tests of significance between means were done by applying Snedecor and Cochran's (6th edition, 1967) revised least significant differences method, i.e. t_{05} or $t_{01} \times MS_{error} (\frac{1}{N_1} + \frac{1}{N_2})$, Df of MS_{error}. Where interaction effects were found in the two-way analysis of covariance, the data were reanalysed in one-way analyses of variance.

This section also considers the differential response of endogenous and non endogenous patients to the three treatment modes. People were classified as endogenous or non-endogenous according to Spitzer's RDC (1978) on the basis of their baseline Hamilton Rating (see Appendix XV for a list of symptoms). X^2 analysis was used to check whether these categories for the two populations combined had a differential response to the three treatments. Fisher's Exact Probability Test was used to compare the frequencies of endogenous responders and non responders and non-endogenous responders and non-responders within each treatment.

1. Analysis of change scores (pre - post treatment)

Table $6B_1$ shows the mean change scores, standard deviations and t values for each treatment in the two populations. Looking at the significance of change scores on all these variables (18) for each treatment ($t = \text{mean} \div \frac{SD}{N}$), the hospital patients improved significantly on nearly all variables in each treatment group. Speech rate (CS) did not change significantly. In addition, for the CBT group there was no significant improvement in outward irritability (OUT) and for drugs the difference between ideal and current self (IDS) did not change significantly. Variables where change would not be expected did not change, i.e. pre-morbid self-esteem (WAS), ideal self (WD), and discrepancy between ideal self and previously well self (IDW). The same pattern applied for general practice combination and CBT groups, i.e. no significant changes for WAS, WD, IDW and CS. However, changes in the general practice drug group were rarely significant. Significant improvement occurred for seven variables only, i.e. depression subscale of the IDA (DEP), and subscales outward irritability (OUT) and total irritability (IRR) as well as view of the self (AM), view of the environment (ENV), ideal self discrepancy (IDS) and discrepancy between pre-morbid self and current self (WASE).

Thus, it can be seen that the three treatment modes were effective for the hospital patients whereas for the general practice patients combination treatment and CBT had an effect on all variables which would be expected to change while drug treatment was, on the whole, not effective.

Table $6B_1$ Mean change scores, standard deviations and t values for each treatment in the two populations

(x, SD's from analysis of covariance)

Number of cases and Df's for each treatment cell in Table $6B_1$

	HP	GP
COMB	N = 13, Df = 12	N = 9, Df = 8
CBT	N = 14, Df = 13	N = 8, Df = 7
D	N = 13, Df = 12	N = 7, Df = 6

Variable	TR	HP		t value		GP		t value	
BDI	COMB	19.8	± 12.7	5.66	***	15	± 7.1	6.33	***
	CBT	10.1	± 14.1	2.68	*	20	± 4.4	12.9	***
	D	15.4	± 9.6	5.77	***	5.4	± 8.3	1.73	N.S.
HS	COMB	6.5	± 7.3	3.2	**	6.7	± 5.6	3.58	**
	CBT	7.3	± 7.5	3.5	**	7.4	± 4.5	4.65	
	D	5.8	± 4.0	5.23	***	3.9	± 8.2	1.26	N.S.
DEP	COMB	4.5	± 4.4	3.69	**	4.6	± 3.4	4.07	**
	CBT	3.6	± 5.0	2.69	*	4.7	± 3.0	4.43	**
	D	4.2	± 3.1	4.88	***	2.0	± 2.1	2.53	*
ANX	COMB	4.8	± 4.4	3.93	**	4.8	± 2.6	5.52	***
	CBT	3.9	± 4.6	3.17	**	4.4	± 1.5	8.30	***
	D	3.1	± 2.8	3.97	**	1.1	± 1.5	1.93	NS
IN	COMB	4.2	± 4.8	3.16	**	3.8	± 2.4	4.75	**
	CBT	3.1	± 4.9	2.37	*	4.5	± 2.7	4.74	**
	D	2.2	± 2.2	3.61	**	0.9	± 2.2	1.08	NS
OUT	COMB	1.9	± 3.4	2.02	NS	2.3	± 2.9	2.37	*
	CBT	1.2	± 2.9	1.54	NS	2.8	± 1.8	4.38	**
	D	0.8	± 1.7	1.70	NS	2.6	± 2.1	3.29	*
IRR	COMB	6.1	± 6.3	3.49	**	6.1	± 4.5	4.07	**
	CBT	4.4	± 7.0	2.35	*	7.3	± 3.7	5.57	***
	D	3.0	± 2.9	3.75	**	3.4	± 3.2	2.81	*
AM	COMB	30.4	± 19.9	5.52	***	24.3	± 12.2	5.97	***
	CBT	19.9	± 22.6	3.29	**	28	± 13.9	5.70	***
	D	20.9	± 11.4	6.61	***	13	± 9.8	3.51	*
WAS	COMB	1.5	± 18.1	.30	N.S.	9.7	± 13.4	2.17	NS
	CBT	6.3	± 19.9	1.18	NS	2.1	± 19.3	0.31	NS
	D	5.2	± 15.7	1.20	NS	7.6	± 17.9	1.13	NS

Variable							
WD	COMB	2.0 ± 3.7	1.96	NS	-.1 ± 3.5	.09	NS
	CBT	2.1 ± 5.5	1.43	NS	1.8 ± 6.8	.99	NS
	D	0.9 ± 9.7	0.33	NS	7.0 ± 13.5	1.38	NS
ENV	COMB	25.5 ± 25.4	3.62	**	30.2 ± 16	5.67	***
	CBT	17.7 ± 18.5	3.58	**	31.4 ± 20.7	4.3	**
	D	15.3 ± 15.1	3.66	**	18.1 ± 19.1	2.5	*
FUT	COMB	33.4 ± 29.4	4.1	**	26.8 ± 12.8	6.28	***
	CBT	21 ± 25.4	3.09	**	20.8 ± 24.9	2.36	*
	D	18.4 ± 21.5	3.09	**	8.4 ± 27.6	0.81	NS
IDS	COMB	32 ± 20	5.7	***	24.2 ± 11.8	6.16	***
	CBT	-2.4 ± 84.8	.11	NS	29.8 ± 10.9	7.74	***
	D	19.2 ± 9.1	7.62	***	20 ± 17.3	3.16	*
IDW	COMB	0.7 ± 18.6	0.14	NS	-9.8 ± 15.1	1.95	NS
	CBT	-4.2 ± 19.2	0.82	NS	-0.4 ± 21.3	.05	NS
	D	-4.2 ± 18.2	0.83	NS	-0.6 ± 11.5	0.14	NS
WASE	COMB	31.8 ± 33.6	3.42	**	34 ± 15.4	6.63	***
	CBT	26.2 ± 35.4	2.77	*	28.4 ± 28	2.88	*
	D	24.9 ± 22.8	3.94	**	22 ± 16	3.64	*
HRS	COMB	13.5 ± 7.5	6.49	***	15.7 ± 5.7	8.26	***
	CBT	9.9 ± 9.2	4.02	**	12.8 ± 3.2	11.33	***
	D	9.3 ± 5.1	6.60	***	3.3 ± 4.8	1.82	NS
CS	COMB	4.4 ± 24.8	0.64	NS	6.9 ± 14.0	1.48	NS
	CBT	18.2 ± 35.9	1.90	NS	11.3 ± 17.3	1.85	NS
	D	-0.5 ± 18.9	0.10	NS	9.3 ± 10.4	2.37	NS
WS	COMB	6.4 ± 10.5	2.2	*	9.0 ± 5.0	5.39	***
	CBT	12.9 ± 13.6	3.5	**	8.6 ± 10.1	2.41	*
	D	11.7 ± 18.1	2.2	*	9.1 ± 11.6	2.08	NS

* $p = < .05$ ** $p = < .01$, *** $p = < .001$

The data from this table were computed in an analysis of covariance for change scores and these are presented in table $6B_2$. There were four co-variates, namely those variables which had discriminated between the two populations at baseline: education level, socio-economic status, duration of illness and total score on the PSE. The

covariates and significance levels for each are shown in Appendix XVI.

Eight variables showed differential effects for treatment, these being BDI, ANX, IN, IRR, AM, ENV, FUT and HRS. There were no location differences and there was only one interaction effect and this was on BDI change scores. Only one of these differences could have occurred by chance at the 5% level and all of these differences were significant at a higher level than the 5% level, except for view of the future (FUT).

Table $6B_2$ Results of analysis of covariance for change scores in the two populations (LOC x TR)

(across group N's: COMB = 22, CBT = 22, D = 20)

Variable	TR	Means, standard deviations, across groups for each TR	Analysis of covariance significance levels	
*	COMB	17.9 ± 10.8	LOC	NS
BDI	CBT	13.7 ± 12.4	TR	$p < .02$
	D	11.9 ± 10.2	LOC x TR	$p < .02$
	COMB	6.5 ± 6.5	LOC	NS
HS	CBT	7.3 ± 6.4	TR	NS
	D	5.1 ± 5.7	LOC x TR	NS
	COMB	4.5 ± 3.9	LOC	NS
DEP	CBT	4.0 ± 4.3	TR	NS
	D	3.4 ± 2.9	LOC x TR	NS
*	COMB	4.8 ± 3.7	LOC	NS
ANX	CBT	4.1 ± 3.8	TR	$p < .01$
	D	2.4 ± 2.6	LOC x TR	NS
*	COMB	4.0 ± 3.9	LOC	NS
IN	CBT	3.6 ± 4.2	TR	$p < .02$
	D	1.8 ± 2.3	LOC x TR	NS
	COMB	2.1 ± 3.1	LOC	NS
OUT	CBT	1.8 ± 2.6	TR	NS
	D	3.2 ± 2.9	LOC x TR	NS
*	COMB	6.1 ± 5.5	LOC	NS
IRR	CBT	5.4 ± 6.1	TR	$p < .05$
	D	3.2 ± 2.9	LOC x TR	NS

Variable	TR	Means, standard deviations, across groups for each TR	Analysis of covariance significance levels	
*	COMB	27.9 ± 17.1	LOC	NS
AM	CBT	22.9 ± 19.9	TR	$p < .01$
	D	18.2 ± 11.3	LOC x TR	NS
	COMB	4.8 ± 16.5	LOC	NS
WAS	CBT	4.8 ± 19.3	TR	NS
	D	6.1 ± 16.0	LOC x TR	NS
	COMB	1.1 ± 3.6	LOC	NS
WD	CBT	1.9 ± 5.9	TR	NS
	D	3.0 ± 11.2	LOC x TR	NS
*	COMB	27.5 ± 21.7	LOC	NS
ENV	CBT	22.7 ± 2.0	TR	$p > .03$
	D	16.3 ± 16.2	LOC x TR	NS
*	COMB	30.7 ± 23.8	LOC	NS
FUT	CBT	20.9 ± 24.6	TR	$p < .05$
	D	14.9 ± 23.6	LOC x TR	NS
	COMB	28.8 ± 16.5	LOC	NS
IDS	CBT	9.3 ± 68.9	TR	NS
	D	19.5 ± 12.2	LOC x TR	NS
	COMB	-3.6 ± 17.7	LOC	NS
IDW	CBT	-2.8 ± 19.6	TR	NS
	D	-2.9 ± 15.9	LOC x TR	NS
	COMB	32.7 ± 27.1	LOC	NS
WASE	CBT	27.0 ± 32.2	TR	NS
	D	23.9 ± 20.3	LOC x TR	NS
*	COMB	14.4 ± 6.7	LOC	NS
HRS	CBT	10.9 ± 7.6	TR	$p < .001$
	D	7.2 ± 5.7	LOC x TR	NS
	COMB	5.4 ± 20.7	LOC	NS
CS	CBT	15.8 ± 30.3	TR	NS
	D	3.3 ± 16.6	LOC x TR	NS
	COMB	7.5 ± 8.6	LOC	NS
WS	CBT	11.3 ± 12.4	TR	NS
	D	10.8 ± 15.7	LOC x TR	NS

* Variables which significantly differentiated the groups at $p < .05$ or less

Figure $6B_2$ shows a histogram of these eight variables for the hospital group. It can be seen that in the hospital group the combination treatment always does best and that there is not much difference between the CBT alone and drug groups. The drug group is usually slightly lower, except for BDI.

Turning to the general practice patients, a histogram of the same variables is shown in figure $6B_3$. In this case, the drug group is always lower in terms of change with only minimal differences between the combination and CBT groups.

Since there was an interaction effect for change scores on the BDI, additional tests for simple main effects were necessary. The results of these are shown in Table $6B_3$.

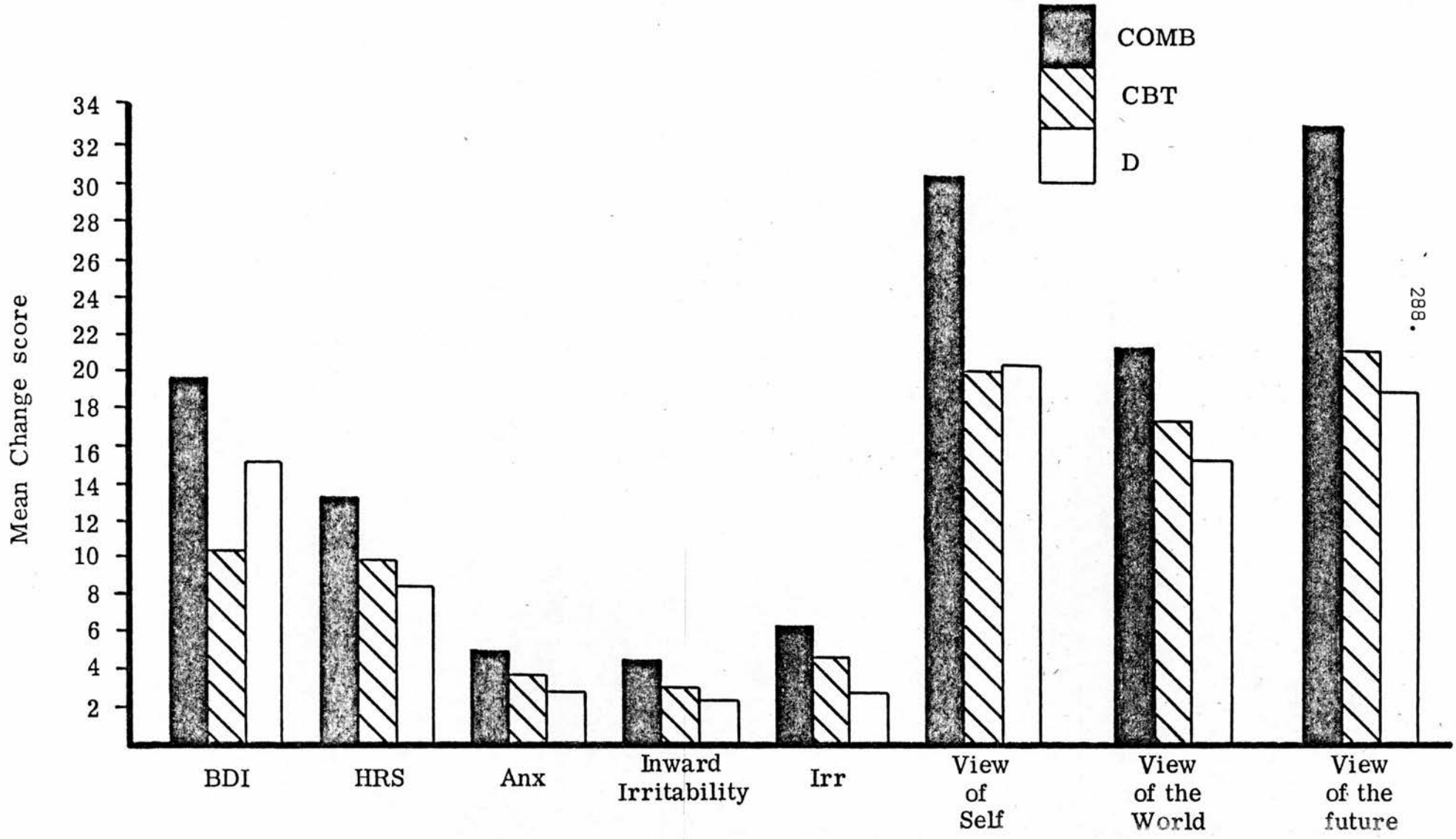
Table $6B_3$ BDI change scores: results of testing for simple main effects using analyses of covariance (1 covariate: DILL)

TR	HP $\bar{x}$, SD	GP $\bar{x}$, SD	Df's	F ratios and significance levels
COMB	19.8 ± 12.7 (N = 13)	15 ± 7.1 (N = 9)	1, 19	F = 29.45 p = < .01
CBT	10.1 ± 14.1 (N = 14)	20 ± 4.43 (N = 8)	1, 19	F = 0.23 NS
D	15.4 ± 9.6 (N = 13)	5.4 ± 8.3 (N = 7)	1, 17	F = 0.08 NS
Df's	2, 36	2, 20	-	-
F ratios and significance levels	F = 4.03 p = < .05	F = 1.11 NS	-	-

Tests between means for the HP

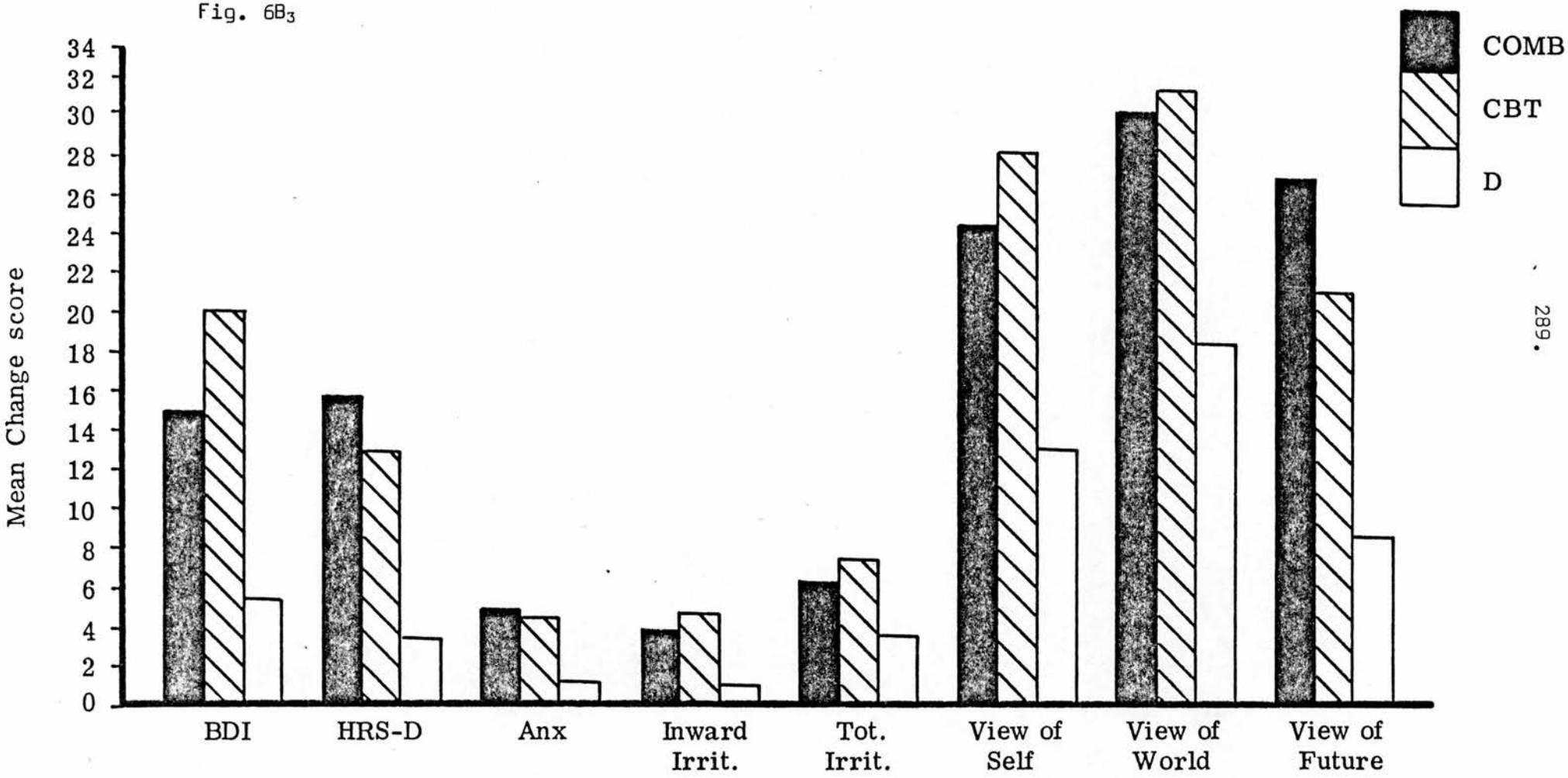
COMB > CBT LSD = 61.7, NS, Df = 36
COMB > D LSD = 65.8, NS, Df = 36
D > CBT LSD = 61.7, NS, Df = 36

Fig. $6B_2$



Mean change scores for hospital group (corrected for DILL, EDUC, SOC, PSE)

Fig. $6B_3$



Mean change scores for the general practice group (corrected for DILL, EDUC, SOC, PSE)

Five one-way analyses of variance were computed for BDI change scores, covarying for duration of illness, this being the only covariate which was significant for this variable. There were no significant differences between locations except for the combination treatment with the hospital combination group doing significantly better than those who received combination treatment in general practice. In treatment effects within locations, the only significant difference was within the hospital group where combination was superior to either treatment on its own. However, no significant differences emerged when a-posteriori tests between means were computed according to the modified LSD method (Snedecor and Cochran, 1967).

Table $6B_4$ Significant results of a-posteriori tests for differences between means in the GP group using the revised LSD method

COMB N = 9 CBT N = 8 Drug N = 7

Variables	TR	$\bar{x}^{GP}$, SD	Comparisons and LSD values
			Df = 53
	COMB	4.8 ± 2.6	COMB > CBT, LSD = 4.2, NS
ANX	CBT	4.4 ± 1.5	COMB > D, LSD = 4.3, *
	D	1.1 ± 1.5	CBT > D, LSD = 4.5, *
			Df = 53
	COMB	3.8 ± 2.4	CBT > COMB, LSD = 3.4, NS
IN	CBT	4.5 ± 2.7	COMB > D, LSD = 3.5, NS
	D	0.9 ± 2.2	CBT > D, LSD = 3.6, *
			Df = 54
	COMB	24.3 ± 12.2	CBT > COMB, LSD = 13.8, NS
AM	CBT	28 ± 13.9	COMB > D, LSD = 14.4, NS
	D	13 ± 9.8	CBT > D, LSD = 14.8, *
			Df = 50
	COMB	15.7 ± 5.7	COMB > CBT, LSD = 6.04, NS
HRS	CBT	12.8 ± 3.2	COMB > D, LSD = 8.4 **
	D	3.3 ± 4.8	CBT > D, LSD = 8.6 **

* $p = < .05$ ** $p = < .01$

N.B. Cases with missing data have been dropped from the analysis which accounts for varying Df's.

Table $6B_4$ shows the other variables which showed significant differences between means when a-posteriori tests were done (LSD method). The only significant differences which emerged were in the general practice on four variables, i.e. anxiety (ANX), inward irritability (IN), view of the self (AM), and the Hamilton Scale (HRS). The differences were between combination therapy and drugs and CBT and drugs but there were no significant differences between combination treatment and CBT.

2. Analysis of patients classified as responders and non-responders

Klerman et al. (1974) defined 'significant clinical improvement' in their pharmacotherapy sample as a 50% decrease in initial depression score (Raskin Scale). This formula was applied in the present study to identify categories of responders and non-responders to the three treatments. Thus, responders were defined as patients who reported at least a 50% reduction from their initial BDI rating and/or a score ≤ 8 on the same measure.

Table $6B_5$ shows the number of responders and non-responders in each treatment for each population. Data from this table were used for six comparisons.

Table $6B_5$ Number of responders/non-responders in each treatment for each population (change in BDI score)

	1 HP			2 GP			Fisher Test Probabilities	
	Resp	Non-resp		Resp	Non-Resp			
COMB	10	3	13	8	1	9	$p = 0.35$	NS
CBT	8	6	14	8	0	8	$p = 0.08$	NS
D	10	3	13	1	6	7	$p = 0.01$	
	28	12	40	17	7	24		

Results of X^2 for differences between responders and non-responders

1. Combined populations, $X^2 = 3.73$, Df = 2, NS
2. Hospital population, $X^2 = 1.70$, Df = 2, NS
3. General Practice $X^2 = 15.81$, Df = 2, $p < .01$

All two-tailed tests of significance

The frequencies of combined responders and non-responders in each treatment for both populations combined was not significant (X^2 = 3.73, Df = 2). Looking at the number of responders and non-responders for each treatment for the hospital population (column 1) X^2 = 1.70, Df = 2, indicating that the frequency of responders and non-responders in each treatment for the hospital patients did not differ significantly. However, for the general practice patients (column 2), X^2 = 15.81, Df = 2, $p = < .01$ which indicated that the number of non-responders in the drug group in this population was disproportionately large.

Looking along the rows at the number of responders and non-responders for each treatment in the hospital and general practice groups, Fisher's exact probability test for combination treatment was p = 0.35 indicating no difference in the frequency of responders and non-responders for this treatment in the two populations. Similarly, there was no difference in cognitive treatment (p = .08), but there was a significant difference in the drug group (p = .01) indicating that in the general practice there were significantly more non-responders to drug than in the hospital group.

3. Analysis of percentage change scores (pre - post treatment)

Table $6B_6$ shows the mean percentage change scores, standard deviations and t values for each treatment in the two populations. Looking at the significance levels of percentage change scores on the 18 variables for each treatment ($t = \text{mean} \div \frac{SD}{N}$), the hospital patients in the combination group improved significantly on 11 variables (BDI, DEP, ANX, OUT, IRR, AM, ENV, FUT, IDS, WASE and HRS). The CBT group improved significantly on 10 variables (BDI, HS, ANX, AM, ENV, FUT, WASE, HRS, CS and WS). For the pharmacotherapy patients

significant improvement occurred on as many variables, these being BDI, HS, DEP, IN, IRR, AM, ENV, IDS, WASE and HRS.

Variables where percentage change would not be expected did not change, i.e. pre-morbid functioning (WAS), ideal self (WD), and discrepancy between ideal self and pre-morbid functioning (IDW). In the combination group hopelessness (HS), inward irritability)IN), speech rate (CS) and writing speed (WS) did not change significantly.

For the CBT group there was no significant improvement in the depression subscale of the IDA (DEP), inward irritability (IN), outward irritability (OUT), total irritability (IRR) and ideal-self discrepancy (IDS). In the hospital pharmacotherapy group, anxiety (ANX), outward irritability (OUT), personal future as assessed by semantic differential (FUT), speech rate (CS) and writing speed (WS) did not change significantly from initial level of severity.

These results indicate that, on the whole, all three treatment modalities were effective in reducing depressive symptoms in the hospital outpatient group. However, when initial differences are taken into account by using percentage change scores, there are fewer significant changes from basal level of depression relative to those found for absolute change scores. For instance, in the present analysis the hospital patients improved significantly in all treatment groups on five variables (BDI, AM, ENV, WASE and HRS). This contrasts markedly with the results of the absolute change analysis where all treatments produced significant improvement on 12 variables in the hospital group.

With respect to the general practice, the same pattern found for absolute change scores applied for percentage change. That is, for the combination and CBT groups there was significant improvement in

all variables, except for personal future (FUT) where there was no significant improvement for the CBT group. Taking initial scores into account, there were even fewer changes in the general practice drug group than in the previous analysis. Significant improvement occurred in only five variables (i.e. DEP, OUT, ENV, IDS and WASE) compared with the seven significantly different variables found in the absolute change score analysis.

Thus, once again, the pharmacotherapy treatment in general practice appears to have had a weak effect in terms of reducing depressive symptoms.

Table $6B_6$ Mean percentage change scores, standard deviations and t values for each treatment in the two populations

(x, SD's from analysis of covariance)

Number of cases and Df's for each treatment cell in table $6B_6$

	HP		GP	
COMB	N = 13	Df = 12	N = 9	Df = 8
CBT	N = 14	Df = 13	N = 8	Df = 7
D	N = 13.	Df = 12	N = 7	Df = 6

Variable	TR	HP	t value	GP	t value
BDI	COMB	70.3 ± 34.5	7.35 ***	70.4 ± 29.8	7.09 ***
	CBT	38.8 ± 65.2	2.23 *	87.8 ± 15	16.58 ***
	D	66.1 ± 38.6	6.18 ***	19.6 ± 31.8	1.63 NS
HS	COMB	23.3 ± 92.2	0.91 NS	64.1 ± 41.2	4.67 **
	CBT	49.1 ± 43.2	4.25 ***	67.9 ± 22.1	8.71 ***
	D	42 ± 26.2	5.79 ***	25.1 ± 58.7	1.13 NS
DEP	COMB	43.4 ± 42.1	3.72 **	45.1 ± 27.1	4.99 **
	CBT	33.1 ± 85.7	1.44 NS	50 ± 28.2	6.10 ***
	D	44.6 ± 24.1	6.68 ***	22.4 ± 20.3	2.92 *
ANX	COMB	45.3 ± 41.1	3.98 **	48.3 ± 21.1	6.87 ***
	CBT	36.5 ± 54.8	2.49 *	50.1 ± 22.5	6.3 ***
	D	17.8 ± 70.1	0.92 NS	12.5 ± 15.9	2.08 NS
IN	COMB	11.3 ± 187.9	0.22 NS	59.4 ± 26.4	6.75 ***
	CBT	29 ± 72.1	1.50 NS	68.3 ± 26.9	7.18 ***
	D	35.1 ± 29.7	4.26 **	18 ± 42.3	1.13 NS

Variable	TR	HP	t value		GP	t value	
	COMB	37.9 ± 56.5	2.42	*	32 ± 39.2	2.45	*
OUT	CBT	11.5 ± 64.3	0.67	NS	38.3 ± 19.2	5.65	***
	D	2.2 ± 40.7	0.20	NS	25.3 ± 23	2.91	*
	COMB	38.6 ± 60.1	2.32	*	47.3 ± 30.8	4.61	**
IRR	CBT	26.5 ± 59.6	1.66	NS	53.4 ± 21.2	7.20	***
	D	24.4 ± 22.1	3.99	**	22.4 ± 24.8	2.39	NS
	COMB	138.7 ±154.9	3.23	**	69.4 ± 51.3	4.06	**
AM	CBT	70.7 ± 82.8	3.19	**	68.7 ± 45	4.32	**
	D	78.1 ± 52.2	5.4	***	37.3 ± 25.2	3.92	**
	COMB	0.5 ± 28.7	.06	NS	14.3 ± 19.6	2.63	*
WAS	CBT	9.0 ± 30.4	1.11	NS	0.1 ± 27.3	0.01	NS
	D	4.5 ± 38.6	0.42	NS	9.1 ± 25.5	0.95	NS
	COMB	2.5 ± 4.6	1.97	NS	0.2 ± 4.6	0.13	NS
WD	CBT	2.6 ± 7.1	1.37	NS	2.5 ± 9.1	0.78	NS
	D	0.4 ± 13.8	0.10	NS	8.6 ± 16.7	1.37	NS
	COMB	152.2 ± 22.2	2.72	*	104 ±103.7	3.01	*
ENV	CBT	52.2 ± 69.6	2.8	*	125 ±155.8	2.27	*
	D	48.4 ± 56	3.12	**	72 ± 71.1	2.68	*
	COMB	200.4 ± 23.3	3.56	**	66.9 ± 36.5	5.5	***
FUT	CBT	77.9 ±103.8	2.81	*	100 ±173	1.64	NS
	D	83.3 ±144.9	2.08	NS	34.8 ± 56	1.64	NS
	COMB	63.6 ± 32.1	7.15	***	61 ± 18.5	9.87	***
IDS	CBT	-5.7 ±182.6	0.12	NS	87.1 ± 28	8.68	***
	D	49 ± 24.6	7.2	***	47.3 ± 36	3.45	*
	COMB	1.2 ± 15.2	0.03	NS	-115.3 ± 179.6	1.93	NS
IDW	CBT	-21.3 ±144.2	0.55	NS	85.5 ±135.8	1.78	NS
	D	-69.6 ±144	1.74	NS	-5.8 ± 90.7	0.16	NS
	COMB	78.3 ± 81.3	3.48	**	160.3 ±216.7	2.22	NS
WASE	CBT	99.5 ±169.4	2.20	*	107.5 ± 57.9	5.25	**
	D	102.7 ±123.9	2.99	**	62.8 ± 45	3.70	**
	COMB	67.7 ± 33.5	7.3	***	73.3 ± 19.2	11.45	***
HRS	CBT	58.2 ± 53.4	4.08	**	79.6 ± 15.8	14.27	***
	D	57.0 ± 29.6	6.95	***	20.3 ± 28.2	1.91	NS
	COMB	-1.8 ± 57.6	0.11	NS	9.2 ± 18.8	1.47	NS
CS	CBT	22.6 ± 29.9	2.83	*	14.6 ± 27.1	1.52	NS
	D	-11.5 ± 56	0.74	NS	12.2 ± 15.4	2.10	NS
	COMB	18.4 ± 36.5	1.82	NS	20.9 ± 18.2	3.44	**
WS	CBT	25.3 ± 27.2	3.48	**	23.5 ± 27.9	2.38	*
	D	222.9 ±700.3	1.15	NS	26.3 ± 33.5	2.07	NS

* $p < .05$, ** $p < .01$ *** $p < .001$

The data from this table were used to compute an analysis of covariance for percentage change scores. These are presented in table $6B_7$. The F ratios, covariates, and significance levels for each are summarised in Appendix XVII.

Table $6B_7$ Results of analysis of covariance for percentage change scores in the two populations (LOC x TR)

(across group N's COMB = 22, CBT = 22, D = 20)

Variable	TR	Means, standard deviations, across groups for each TR	Analysis of covariance significance levels	
*	COMB	70.3 ± 34.5	LOC	NS
BDI	CBT	56.7 ± 57.4	TR	$p < .04$
	D	49.8 ± 42.2	LOC x TR	$p < .01$
	COMB	40 ± 77	LOC	NS
HS	CBT	56.3 ± 37.1	TR	NS
	D	36.1 ± 39.8	LOC x TR	NS
	COMB	44.1 ± 36	LOC	NS
DEP	CBT	39.3 ± 69.2	TR	NS
	D	36.8 ± 24.8	LOC x TR	NS
*	COMB	46.5 ± 33.7	LOC	NS
ANX	CBT	41.4 ± 45.5	TR	$p < .01$
	D	15.9 ± 56.5	LOC x TR	NS
	COMB	30 ± 14.5	LOC	NS
IN	CBT	43.3 ± 61.9	TR	NS
	D	29.1 ± 34.5	LOC x TR	NS
	COMB	35.5 ± 49.2	LOC	NS
OUT	CBT	21.2 ± 53.5	TR	NS
	D	10.3 ± 36.6	LOC x TR	NS
	COMB	42.2 ± 49.5	LOC	NS
IRR	CBT	36.4 ± 50.3	TR	NS
	D	23.7 ± 22.4	LOC x TR	NS
*	COMB	110.4 ±126.2	LOC	NS
AM	CBT	70 ± 70.1	TR	$p < .04$
	D	63.8 ± 48.2	LOC x TR	NS
	COMB	5.6 ± 25.9	LOC	NS
WAS	CBT	5.8 ± 27	TR	NS
	D	6.1 ± 33.9	LOC x TR	NS
	COMB	1.4 ± 4.7	LOC	NS
WD	CBT	2.6 ± 7.7	TR	NS
	D	3.3 ± 15	LOC x TR	NS

Variable	TR	Means, standard deviations, across groups for each TR	Analysis of covariance significance levels	
	COMB	132.5 ± 167.3	LOC	NS
ENV	CBT	78.7 ± 111.2	TR	NS
	D	56.7 ± 60.9	LOC x TR	NS
	COMB	145.8 ± 169	LOC	NS
FUT	CBT	86 ± 129.4	TR	NS
	D	66 ± 121.7	LOC x TR	NS
	COMB	62.5 ± 26.9	LOC	NS
IDS	CBT	28.1 ± 151.6	TR	NS
	D	48.4 ± 28.3	LOC x TR	NS
	COMB	-46.5 ± 170.1	LOC	NS
IDW	CBT	17.6 ± 147.6	TR	NS
	D	-47.1 ± 129.4	LOC x TR	NS
	COMB	111.9 ± 152.9	LOC	NS
WASE	CBT	102.4 ± 137.4	TR	NS
	D	88.7 ± 103.5	LOC x TR	NS
*	COMB	70 ± 28.1	LOC	NS
HRS	CBT	66 ± 44.3	TR	$p < .01$
	D	44.2 ± 33.6	LOC x TR	NS
	COMB	2.9 ± 45.4	LOC	NS
CS	CBT	19.8 ± 28.5	TR	NS
	D	-2.3 ± 45.5	LOC x TR	NS
	COMB	19.4 ± 29.8	LOC	NS
WS	CBT	24.7 ± 26.8	TR	NS
	D	154.1 ± 56.5	LOC x TR	NS

* Variables which significantly differentiated the groups at $p = < .05$ or less

Controlling for baseline levels of severity resulted in four differential effects for treatment, i.e. self-reported depression (BDI), anxiety (ANX), view of current self (AM) and observed depression (HRS). One of these could have occurred by chance at the 5% level.

In contrast, analyses of change scores irrespective of initial level of severity produced eight significant treatment effects on variables where change would have been expected. As in the change score analysis, there was an interaction effect for BDI percentage change.

Figure $6B_7$ shows a histogram of the four variables which showed differential effects for treatment in both populations. It can be seen that in the hospital group the combination treatment continues to do best and that there is not much difference between the CBT and drug groups. The drug group is slightly lower for observed depression (HRS) and anxiety (ANX) but slightly higher than CBT on the cognitive measure of current self esteem (AM). For the BDI, the drug group responds better than the CBT group and almost to the same level as the combination therapy group.

For the general practice patients, figure $6B_7$ shows that once again the drug group is lowest with minimal differences between the combination and CBT groups.

The results of the additional tests for simple main effects for the BDI are presented in table $6B_8$.

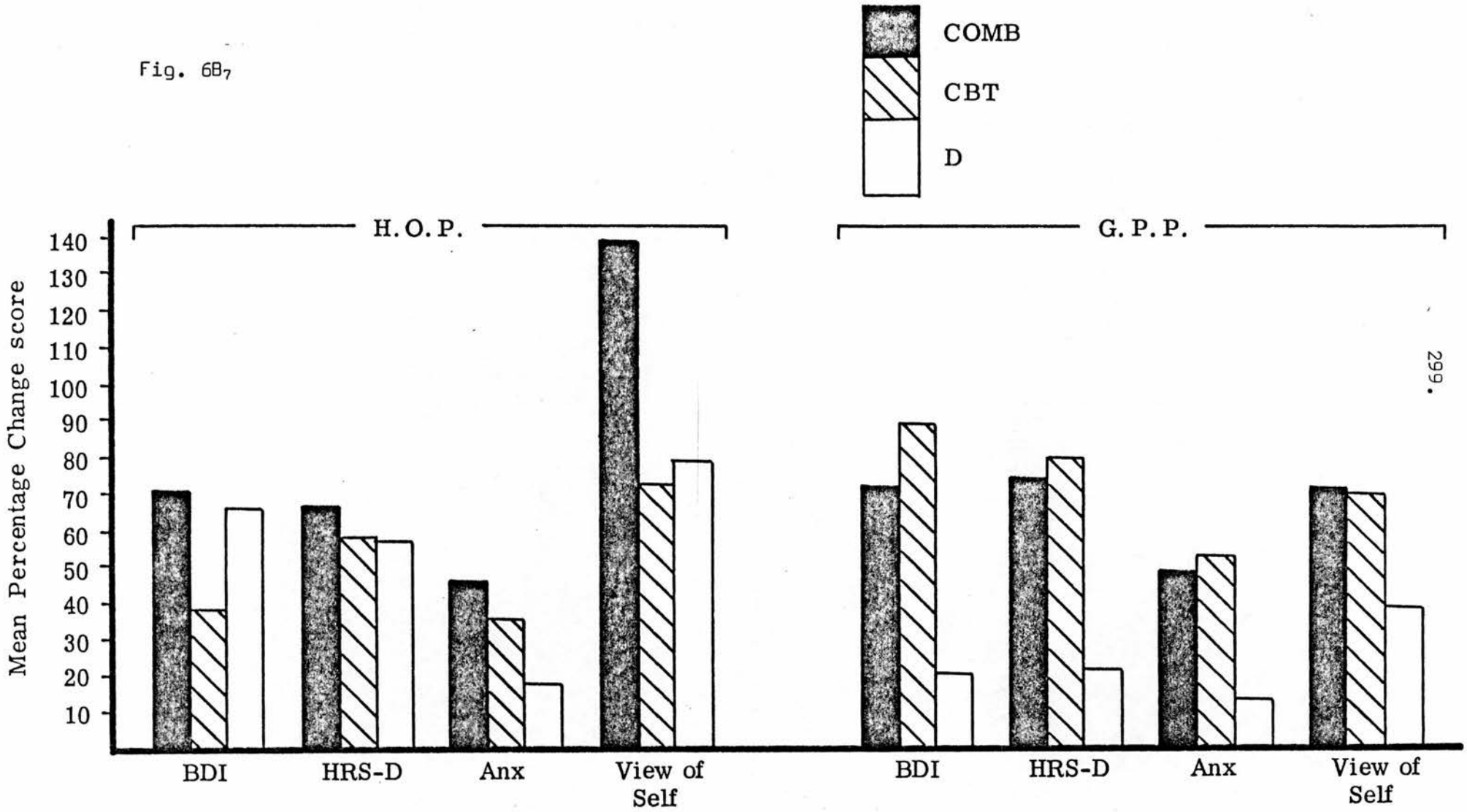
Table $6B_8$ BDI percentage change scores: results of testing for simple main effects using analyses of covariance

(1 covariate)

	$\bar{x}$ HP, SD	$\bar{x}$ GP, SD	Df's	F ratios and significance levels
COMB	70.3 ± 38.5 (N = 13)	70.4 ± 29.8 (N = 9)	1, 19	F = 13.65 p = < .01
CBT	38.8 ± 65.2 (N = 14)	87.9 ± 15 (N = 8)	1, 19	F = .16 NS
D	66.1 ± 38.6 (N = 13)	19.6 ± 31.8 (N = 7)	1, 17	F = .31 NS
Dfs	2, 36	2, 20	-	-
F ratios and significance levels	F = 2.93 NS	F = 1.11 NS	-	-

Five one-way analyses of variance were computed for BDI percentage change scores covarying for duration of illness. Although the mean percentage changes were nearly identical, there was a highly significant difference between locations for the combination treatment.

Fig. $6B_7$



Mean percentage change scores for both populations (corrected for DILL, EDUC, SOC, PSE)

It will be remembered that the hospital patients were significantly more chronic than the general practice patients. When the effects of duration of illness are controlled, it appears that the hospital combination group does significantly better than the general practice combination group. Similarly, when the effects of chronic depression are partialled out in the other comparisons, no significant differences emerge either between or within locations.

Table $6B_9$ shows the results of the tests of significance for differences between means for the HRS. The only significant differences which emerged were in the general practice group. These were between combination and drugs and CBT and drugs and there was no difference between combination and CBT. Thus, cognitive therapy with or without drugs did better than drugs alone in the general practice. This was true for observed depression but not the self-reported aspects of depression which did, in fact, show differential treatment effects in the two-way analysis of covariance.

Table $6B_9$ HRS percentage change score: a-posteriori tests for differences between means in the GP group using the revised LSD method

COMB N = 9		CBT N = 8	Drug N = 7
Variable	TR	GP	Comparisons and LSD values Df = 50
HRS	COMB	73.3 ± 19.2	CBT > COMB, LSD = 32.8, NS
	CBT	79.6 ± 15.8	COMB > D, LSD = 34, p <.01
	D	20.3 ± 28.2	CBT > D, LSD = 34.8 p <.01

4. The differential response of endogenous and non-endogenous patients to the three treatments

Since many clinicians assume that endogenous depressed patients respond to drug therapy and non-endogenous patients are perhaps best

suited for psychological interventions, it was thought desirable to investigate the differential response of these subgroups to the three treatment modes. Thus, patients were classified as definite endogenous or non-endogenous according to Spitzer's RDC (for a list of symptoms and the criteria for inclusion, see Appendix XV). This was done in a post hoc fashion on the basis of baseline Hamilton Ratings. The hospital and general practice patients were combined for this analysis.

From table $6B_{10}$ it can be seen that the proportion of endogenous and non-endogenous patients were equally distributed across the three treatments.

Table $6B_{10}$ Frequencies of endogenous and non-endogenous patients in each treatment across populations

TR	Endogenous	Non-endogenous	Total
COMB	11	11	22
CBT	7	15	22
D	9	11	20
Total	27	37	64

$x^2 = 1.07$, Df = 2, NS
(Two tailed test)

The frequencies of endogenous responders and nonresponders and non-endogenous responders and non-responders within each treatment are presented in table $6B_{11}$. Along the rows, comparing these categories by Fisher's Exact Probability Test, there were no significant differences (2 x 2 tables).

Comparing the first two columns, i.e. the frequencies of responders and non-responders to the three treatments within the endogenous group,

there was no significant difference according to x^2 analysis (3 x 2 table). Similarly, comparing the last two columns, there was no difference in the response of the non-endogenous group.

Table $6B_{11}$ Response of endogenous and non-endogenous subgroups in the two populations combined

TR	Endogenous (N = 27)		Non-endogenous (N = 37)		Fisher Tests
	Responders	Nonresponders	Responders	Non-responders	
COMB	9	2	9	2	1.42, NS
CBT	5	2	11	4	1.44, NS
D	5	4	6	5	1.30, NS
	x^2 = .70, NS		x^2 = .89, NS		

(Two-tailed tests)

Thus, in this group, endogenous patients respond as well to CBT as they do to drugs or combination treatment and non-endogenous patients respond as well to drugs as they do to CBT with or without drugs.

5. Pattern of response in the subgroup of responders

In theory the three treatment modes could be said to exert their effects at different or multiple levels of functioning, i.e. drugs at the biological level, CBT at the cognitive or psychological level, and combination therapy at both levels simultaneously. The end result for patients in each group seems to be at least partial recovery from depression in terms of mean change or percentage change scores. But what about the degree of response to the different treatments at various stages of therapy? If these treatments do exert effects at different

or multiple levels of functioning then they might be expected to produce different patterns of response over time. Moreover, differential patterns of response might be most apparent in subgroups of patients who respond well to each treatment. By looking at the pattern of response for each treatment in groups of responders, it may be possible to identify the point in time at which patients may be expected to show significant improvement in the different treatments.

The next section, while essentially descriptive, attempts to consider these questions. Nonresponders were excluded from the analysis because the main interest here was to delineate the 'typical' sort of response which might be expected from patients who respond to combination therapy, CBT, or drugs.

The only analysis used was a series of post hoc tests (t tests) to compare 1) the degree of response at consecutive points in time for each treatment on its own, and 2) the degree of response at consecutive points in time across the three treatments. This analysis, it was thought, might give some idea as to when improvement might be expected for each treatment and which treatment produced the quickest response. The analysis was computed for self-rated depression (BDI) and hopelessness (HS) only.

Ordinarily, when examining data over time, particularly change scores, a repeated measures analysis of variance would be the statistical method of choice (see Winer, 1970). However, the population studied was, by definition, a highly selected subgroup known to have shown improvement, i.e. BDI final score $< \underline{8}$ or 50% reduction from initial score. Therefore, it was considered unnecessary to do an analysis of variance for repeated measures and quite valid to make the various comparisons using post hoc tests.

Figure $6B_8$ shows the pattern of response in groups of responders to each treatment (two samples combined) on self-rated depression (BDI). Inspection of the graph shows no differences at initial assessment and no difference at outcome but the pattern of response appears different for the three treatments. Pharmaco-therapy seems to provide a very stable response over time. In contrast, cognitive therapy gives a dramatic response initially but is, on the whole, more variable. Combination treatment appears to combine stability with quickness of response.

The results of paired t-tests of mean differences in BDI change scores between consecutive occasions of testing within treatments are presented in table $6B_{12}$. For the drug group, there are no significant changes between any two consecutive points in time, except for T_5–T_6, which suggests a very steady, gradual reduction of depression throughout the course of treatment (approximately two weeks between assessments, 14 weeks altogether). The first significant improvement from baseline assessment occurs at T_3, or approximately six weeks into therapy. On the other hand, cognitive therapy gives a highly significant rate of improvement at approximately two weeks into treatment and, following a more erratic course, shows less significant changes between times 3 and 5, or 6 to 10 weeks into treatment. In fact, the pattern shown at the early stages for cognitive therapy appears similar to the placebo effect discussed by Frankel (1978). For combination treatment, there is also an immediate reduction in level of depression from baseline to time 2 and another significant change in the subsequent two weeks, though there is no significant change between any two points in time afterwards.

Fig. $6B_8$

Pattern of response for each treatment (2 samples combined)
Responders only - self-rated depression

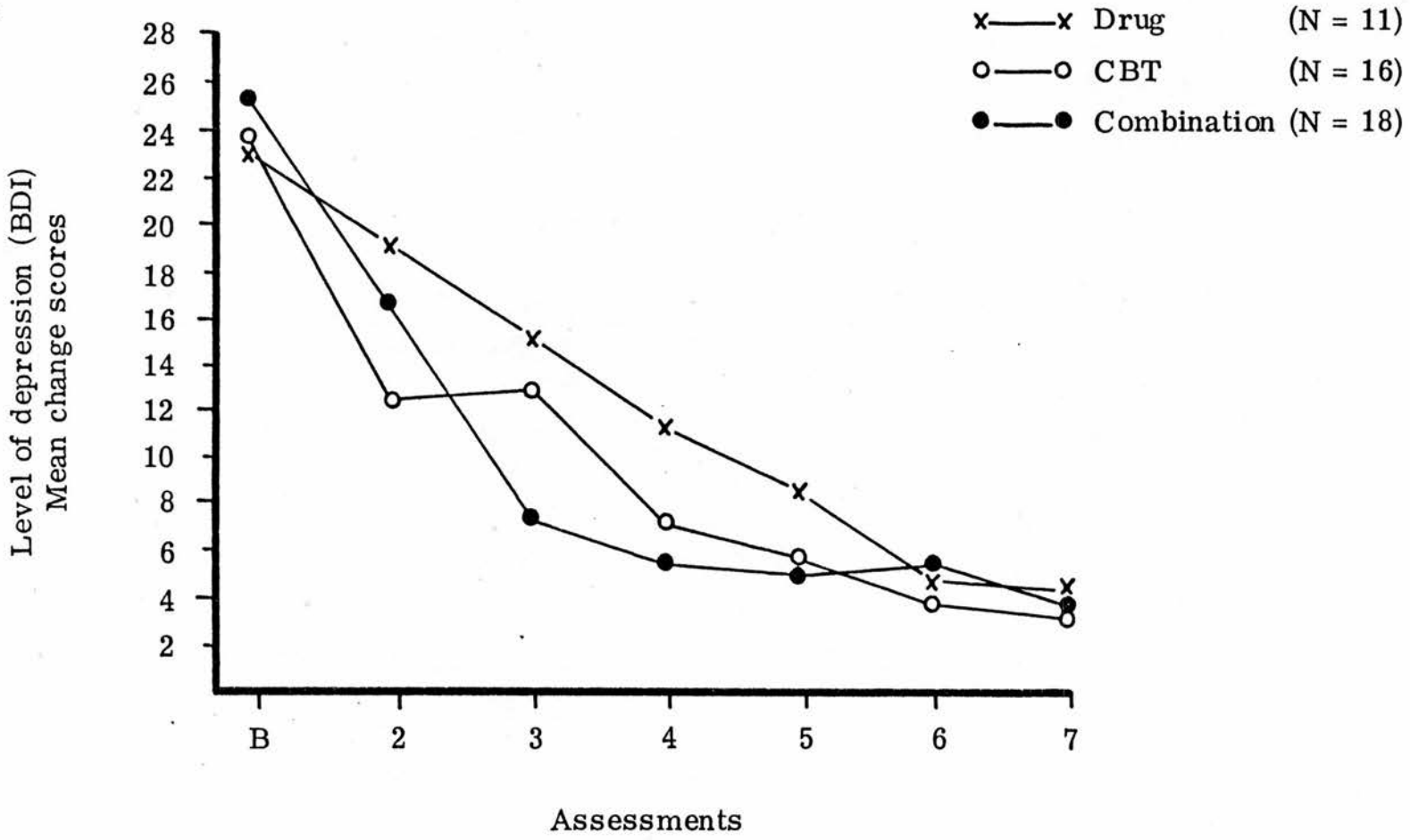


Table $6B_{12}$ Results of t-tests of mean differences in BDI change scores between consecutive occasions of testing: within treatments

Responders only

Treatment	B – T_2	T_2 – T_3	T_3 – T_4	T_4 – T_5	T_5 – T_6	T_6 – T_7	Time of first significant change
Drug (N = 11) Df = 10	t = 1.72 NS	t = 1.59 NS	t = 1.69 NS	t = 1.58 NS	t = 2.3*	t = .37 NS	B – T_3 t = 3.37**
CBT (N = 16) Df = 15	t = 6.25***	t = .11 NS	t = 3.53**	t = 2.37*	t = .65 NS	t = .75 NS	B – T_2
COMB (N = 18) Df = 17	t = 5.62***	t = 3.42**	t = 1.98 NS	t = .72 NS	t = .60 NS	t = 1.19 NS	B – T_2

* p = < .05 ** p = < .01 *** p = < .001

Table $6B_{13}$ shows the results of t-tests of mean differences in BDI change scores between occasions of testing across the three treatments. There were no differences at initial assessment, though there are significant differences at the 3rd and 4th assessments, i.e. at 6 and 8 weeks approximately. The differences on each of these occasions are between pharmacotherapy and combination treatment, with the latter providing the quicker response. There were no significant differences between cognitive therapy and combination treatment.

Table $6B_{13}$ Results of t-tests of mean differences in BDI change scores between consecutive occasions of testing: across treatments

	Drug N = 11		CBT N = 16		COMB N = 18		
Treatment Comparisons	Base-line (B)	T_2	T_3	T_4	T_5	T_6	T_7
D v COMB Df = 27	t = .85 NS	t = .88 NS	t = 2.77 **	t = 2.38 *	t = 1.36 NS	t = .11 NS	t = .67 NS
D v CBT Df = 25	t = .49 NS	t = 1.90 NS	t = .79 NS	t = 1.56 NS	t = 1.95 NS	t = .63 NS	t = .88 NS
COMB v CBT Df = 32	t = .78 NS	t = 1.30 NS	t = 1.97 NS	t = .60 NS	t = .71 NS	t = .46 NS	t = 1.2 NS

* $p = < .05$ ** $p = < .01$

The differential response rate of the three treatments is shown more dramatically on hopelessness (Figure $6B_9$). Though there seem to be no differences at baseline, from the 3rd assessment there appears to be large differences between the drug responders and the responders from the other treatments and these are evident even at the end of treatment.

Table $6B_{14}$ shows the results of paired t-tests of mean differences on hopelessness change scores between consecutive occasions of testing within each treatment. With regard to the drug group, there were no

Fig. 6B9

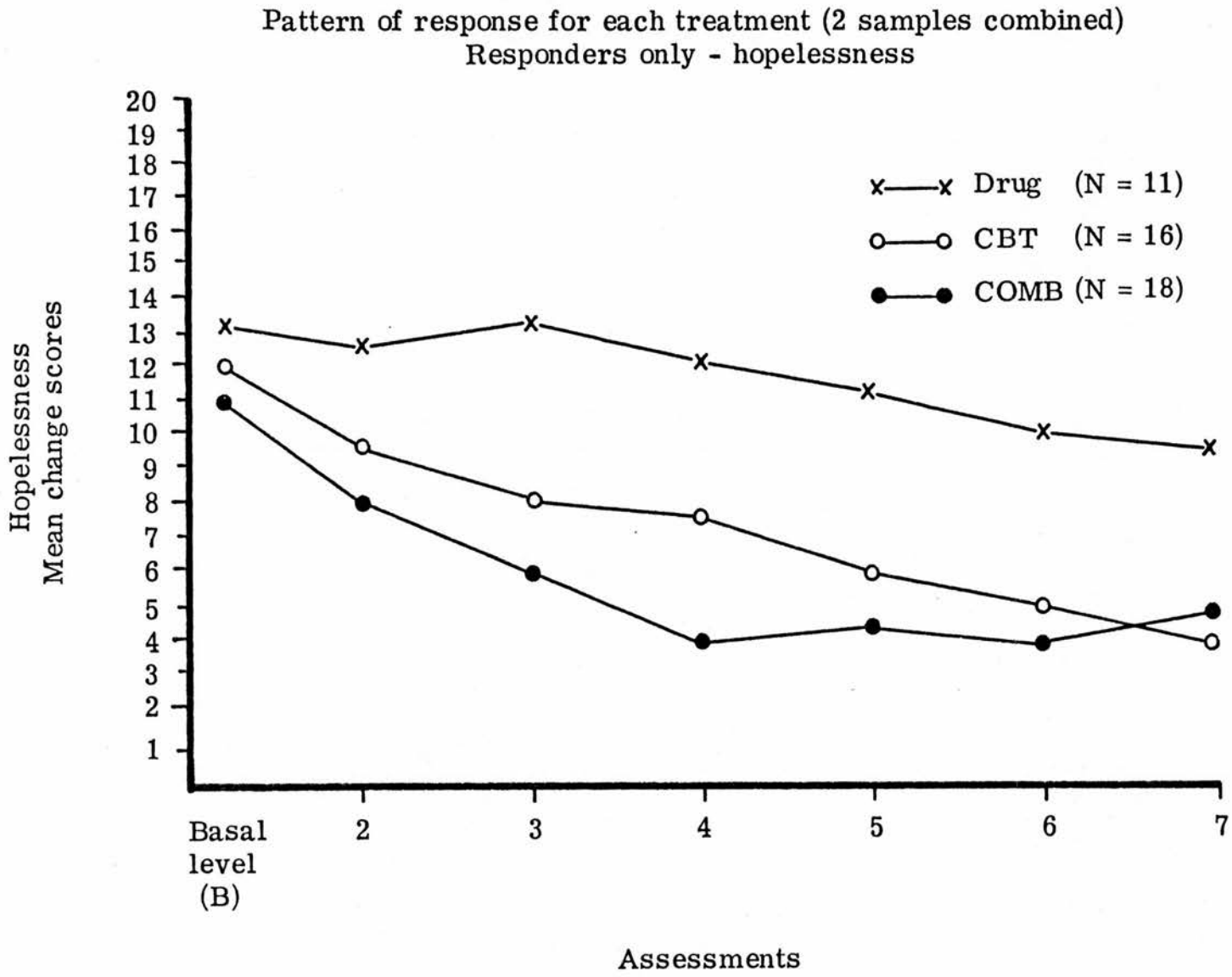

Pattern of response for each treatment (2 samples combined)
Responders only - hopelessness
Hopelessness
Mean change scores
20
19
18
17
16
15
14
13
12
11
10
9
8
7
6
5
4
3
2
1
Drug (N = 11)
CBT (N = 16)
COMB (N = 18)
Basal level (B)
2
3
4
5
6
7
Assessments

Table $6B_{14}$ Results of t-tests of mean differences in HS change scores between consecutive occasions of testing: within treatment

Responders only

Treatment	B – T_2	T_2 – T_3	T_3 – T_4	T_4 – T_5	T_5 – T_6	T_6 – T_7	Time of first significant change
Drug (N = 11) Df = 10	t = .55 NS	t = .74 NS	t = 1.95 NS	t = 1.36 NS	t = 1.25 NS	t = .45 NS	B – T_5 t = 3.03 *
CBT (N = 16) Df = 15	t = 2.87*	t = 1.24 NS	t = 2.0 NS	t = 1.78 NS	t = .23 NS	t = .48 NS	B – T_2
COMB (N = 18) Df = 17	t = 2.42*	t = 3.24**	t = 1.47 NS	t = .13 NS	t = .28 NS	t = .78 NS	B – T_2

* p = < .05 ** p = < .01

significant changes between any two consecutive points in time. The rate of change for drugs is slow. Significant improvement does occur in this group of responders but not until approximately 10 weeks into treatment (T_5). In contrast, cognitive therapy gives a significant rate of improvement at approximately two weeks into therapy. Moreover, there are no significant changes between the remaining occasions of testing which suggests a steady, gradual reduction of hopelessness over time. This stable pattern contrasts sharply with the more erratic response pattern seen for cognitive therapy on self-rated level of depression. For combination treatment, there is an immediate and significant improvement in the first two weeks (T_2) followed by another and more highly significant reduction in hopelessness between weeks two and six ($T_2 - T_3$). Overall, the pattern for combination appears similar to that for cognitive therapy and both of these are markedly different from the drug therapy response pattern.

The results of t-tests of mean differences in hopelessness across the three treatments at the different points in time are shown in Table $6B_{15}$. There are no significant differences at baseline. There are, however, highly significant differences between the drug and combination treatments from T_3 onwards until the end of treatment. Also, there are significant differences across the same points in time between drugs and cognitive therapy. In both sets of comparisons, cognitive therapy with or without drugs produced the quickest reduction in hopelessness. However, when cognitive therapy and combination treatment are compared over the different occasions of testing, there are no significant differences, indicating an equivalent effect for the two treatments on hopelessness.

Table $6B_{15}$ Results of t-tests of mean differences in HS change scores between consecutive occasions of testing: across treatments

Responders only

Treatment comparison	Baseline	T_2	T_3	T_4	T_5	T_6	T_7
D v COMB Df = 27	t = .93 NS	t = 1.46 NS	t = 4.51***	t = 4.29***	t = 3.26**	t = 3.38**	t = 2.82**
D v CBT Df = 25	t = .43 NS	t = 1.53 NS	t = 2.41*	t = 2.28*	t = 3.50**	t = 3.45**	t = 4.34***
COMB v CBT Df = 32	t = .46 NS	t = .57 NS	t = 1.70 NS	t = 1.57 NS	t = .05 NS	t = .13 NS	t = .47 NS

* p = <.05 ** p = < .01 *** p = < .001

C. PREDICTION OF RESPONSE TO THERAPY: MULTIPLE REGRESSION ANALYSIS

An important but secondary aim of this investigation was to elicit predictive therapy and patient factors. To accomplish this, different statistical techniques were used, namely multiple regression and discriminant function analyses respectively. This part of the results is concerned with multiple regression, the method used to arrive at the optimum combination of predictor variables for each treatment group. The criterion measure selected was percentage change on the main self-report scale for level of depression (BDI).

Multiple regression represents the maximum correlation between a criterion or dependent variable and a weighted combination of independent variables and is said to give an inflated value because it capitalises upon chance deviations that favour high multiple correlations (e.g. Draper-Smith, 1966). Some have argued that it is a biased estimate of multiple correlation in the population and that this is especially true when the sample sizes are small as they are in this study (for detailed discussion see Kerlinguer, 1973; Draper-Smith, 1966). The statistical technique used to make this computation computes an adjusted R^2 which takes account of both the number of variables in the equation and the number of cases and is designed to adjust the significance of R^2 when sample sizes are small. The procedure used was a stepwise regression which is described fully in the Manual of the Statistical Package for the Social Sciences (SPSS, 2nd edition, 1975).

Using percentage changes on the BDI as the criterion measure, most of the other variables were included as predictor variables in three multiple regression analyses, one for each treatment (both populations

combined). Six variables were excluded from the analyses in order to minimise the amount of redundant information in the regression equations and to enhance clinical credibility. These were DEP, IN, OUT, CS, WS and SOC. The subscales of the IDA (DEP, IN, OUT) were excluded because it was felt that these measures would be unlikely to contribute any more valid information than, for example, the Hamilton Scale for Depression (HRS) and the measure of total irritability (IRR) which, in fact, is simply a composite of the other irritability subscales. The behavioural measures (CS, WS) were also excluded partly because they proved to be among the least sensitive measures of severity of depression (see section A, para. 5) and partly because relative to other measures (e.g. mood and cognitive), they appeared to be less meaningful from a clinical standpoint. Since social class (SOC) was found to be highly correlated with education, it was considered unnecessary to include both variables in the same analyses. Level of education was thought to be more relevant in view of the possible relationship between response to psychotherapy and verbal skills (Whitehead, 1979).

Thus, a total of 19 variables were used in the regression equations for the three treatments. These were: location (LOC), age, sex, education (EDUC), duration of illness (DILL), PSE total severity score (PSE), Beck Depression Inventory (BDI), Anxiety (ANX), total irritability (IRR), Hamilton Rating Scale (HRS), hopelessness (HS), Me as I am now (AM), Me as I was before this illness (WAS), Me as I would like to be (WD), My environment as it is now (ENV), the Future as I see it for me (FUT), discrepancy between ideal and current self(IDS), discrepancy between ideal and premorbid self (IDW) and discrepancy between previous and current self (WASE).

This section presents the results of the stepwise multiple regression. Although all of the variables were included in the analyses, only the 'best' predictor variables for each treatment are reported here, i.e. the fewest number of variables which, in combination, accounted for the greatest proportion of variation in percentage change on self-rated depression. The maximum proportion of variance explained in the various regression analyses using all 19 variables was 70 per cent.

Once the optimum combination of predictor variables had been obtained, those variables with significant regression coefficients were used to devise three equations which were subsequently employed to arrive at 'predicted groups' for combination, CBT and drugs respectively.

Table $6C_1$ shows the best predictors of percentage change in self-rated depression for combination treatment. Looking at the R^2 column, six variables account for 59% of the variation in the criterion measure.

Table $6C_1$ Stepwise multiple regression analyses: combination therapy Summary of the best predictors of percentage change in self-rated depression (BDI)

Variables added	Simple R	Multiple R	R^2	R^2 change
DILL	-0.48	0.47	0.23	0.23
EDUC	0.12	0.67	0.46	0.23
IRR	-0.24	0.69	0.49	0.03
ANX	0.16	0.72	0.52	0.03
WASE	-0.07	0.73	0.53	0.01
AM	0.27	0.76	0.59	0.06

Of these duration of illness (DILL) and level of education (EDUC) explain 46% of the variance when combined in the regression analysis. The direction and strength of the relationship between these two predictors

and change in self-reported depression can be seen in table $6C_2$, which shows the unstandardised (B) and standardised (Beta) regression coefficients for duration of illness and educational level. For combination treatment long duration of illness is a negative predictor (B = -0.37, Beta = -0.810) and high level of education is a positive predictor (B = + 17.8, Beta + 0.580) of percentage change in self-rated depression. The F tests for each regression coefficient (B's) are significant, indicating that there is a direct linkage between these two variables and outcome as assessed by percentage change in the BDI. Table $6C_2$ also shows the two variable regression equations for the combination group.

Table $6C_2$ Stepwise multiple regression analysis

Two variable equation for the combination group with % change in BDI as the criterion (R = +0.67, adjusted R^2 = +0.40)

Variable	B	Beta	Df	F ratio
DILL - duration of illness	-0.37	-0.810	1, 19	15.26 **
EDUC - eduational level	17.8	+0.580	1, 19	7.85 *

(Constant 65)

Regression equation for the combination group

Combination = 65 - 0.37 (DILL) + 17.8 (EDUC)

** p = < .01 * p = < .05

A summary of the best predictors of percentage change in BDI for CBT is presented in table $6C_3$. The column R^2 indicates that four variables account for 53% of the variation in the criterion measure,

Table $6C_3$ Stepwise multiple regression analysis: CBT

Summary of the best predictors of percentage change in self-rated depression (BDI)

Variables added	Simple R	Multiple R	R^2	R^2 change
DILL	-0.55	0.55	0.30	0.30
ENV	-0.37	0.67	0.45	0.15
BDI	0.45	0.70	0.50	0.05
LOC	0.39	0.73	0.53	0.03

with duration of illness (DILL) and view of the environment (ENV) contributing as much as 45% of the variance. The unstandardised (B) and standardised (Beta) regression coefficients for these two predictors are shown in Table $6C_4$, giving an idea as to the direction and strength of the association between duration of illness, view of the environment and percentage change in BDI. For cognitive therapy, again long duration of illness is a negative predictor (B = -0.29, Beta -.558) as is a relatively positive view of the environment (B = -1.36, Beta = -0.389), i.e. patients with a negative view of the environment (low scores) tend to do better on cognitive therapy. There is a direct association between these two variables and outcome on self-rated depression as evidenced by the significant F ratios for each regression coefficient (B's) computed in the analysis. Table $6C_4$ also shows the two variable equation for the CBT group.

Table $6C_4$ Stepwise multiple regression analysis: two variable equation for the CBT group with % change in BDI as the criterion ($R = +0.67$, adjusted $R^2 = +0.39$) N = 22

Variable	B	Beta	Df	F ratio
DILL - duration of illness	-0.29	-0.558	1, 19	10.21**
ENV - view of the environment	-1.36	-0.389	1, 19	4.97 *

Regression equation for the CBT group

cognitive therapy alone = 147.66 - 0.29 (DILL) - 1.36 (ENV)

** p = < .01 * p = < .05

Table $6C_5$ shows the best predictors of percentage change in self-rated depression for drug therapy. Inspection of column R^2 indicates that four variables account for 56% of the variance. Of these referral source (LOC) and perceived discrepancy between ideal and current self (IDS) combined explain 47% of the variation in the criterion measure.

Table $6C_5$ Stepwise multiple regression analysis: drug treatment
Summary of the best predictors of percentage change in self-rated depression (BDI)

Variables added	Simple R	Multiple R	R^2	R^2 change
LOC	-0.54	0.54	0.29	0.29
IDS	-0.45	0.69	0.47	0.18
ANX	-0.39	0.72	0.52	0.05
HS	0.06	0.95	0.56	0.04

The direction and strength of the relationship between these two predictors and change in self-rated depression can be seen in table $6C_6$, which shows the

unstandardised (B) and standardised (Beta) regression coefficients for referral source and perceived discrepancy between ideal and current self. For drug therapy, being a patient attending a general practice in a predominently working class area is a negative predictor (B = -.45, Beta = -0.527) and having a large discrepancy between current self-image and ideal self is also a negative predictor. The F tests for each regression coefficient (B's) are significant, suggesting a direct relationship between these two variables and outcome as measured by percentage change in BDI. The two variable equation for the drug therapy group is also shown in table $6C_6$.

Table $6C_6$ Stepwise multiple regression analysis

Two variable equation for the drug groups with % change in BDI as the criterion ($R = +0.69$, adjusted $R^2 = +0.42$) $N = 20$

	B	Beta	Df	F ratio	
LOC - source of referral	-.45	-0.527	1, 17	9.06	**
IDS - perceived discrepancy between ideal and current self	-1.2	-0.43	1, 17	6.07	*

(Constant = 165.0)

Regression equation for the drug group

Drugs = 165 - 45 (LOC) - 1.2 (ideal-self discrepancy)

** $p = < .01$ * $p = < .05$

D. SUMMARY OF RESULTS

1. Patients from both sources of referral reported substantial psychopathology as indicated by pattern of symptoms, total severity score on the PSE, self-reported depth of depression and observer rated depression. Although the two samples were similar in terms of pattern of affective disturbance as assessed on the PSE, the hospital group were more severely disturbed than the general practice patients. Moreover, the hospital sample reported longer duration of current depressive episode than the community based sample of patients.

2. Attrition was considerably higher in the general practice group but the difference was not significant. There were no differences in the attrition rate across treatments in each population.

Patients who dropped out of treatment were more depressed on self-rated and observer rated depression, more inwardly irritable and generally irritable, and more negative in their perception of the environment compared to those who remained in treatment.

3. Hospital patients were better educated and from higher social class than general practice patients.

4. In the analysis of simple change scores within treatments, all treatment groups in the hospital sample improved on most of the dependent measures, as did patients in both cognitive therapy groups in the general practice sample. In contrast, changes in the general practice drug group were relatively rare.

5. Analysis of covariance for change scores indicated differential effects for treatment on five mood measures and the three cognitive measures pertinent to Beck's theory. For the hospital group, combination treatment produced the greatest improvement, followed by CBT and drugs. After disentangling the interaction effects for self-reported depression (BDI), the pattern was: combination, then pharmacotherapy and CBT. For the general practice group, the drug treatment always showed the least improvement compared to changes in both of the cognitive therapy groups.

6. With regard to percentage change scores, all three treatments were effective in reducing depressive symptoms in the hospital group,

though there were fewer significant changes compared to the number of changes found in the analysis of absolute change scores. For the general practice patients, combination and cognitive therapy alone resulted in significant improvement in depressive symptoms. In contrast, the drug group again showed the least number of significant changes and even fewer changes than were found for this group in the previous analysis.

7. Differential response rates for the three treatments in each population for percentage change scores were similar to those found in the analysis of absolute change scores. Again, controlling for initial differences on basal measures resulted in fewer variables which showed differential treatment effects, i.e. three mood measures and one cognitive measure.

8. Correcting for duration of illness markedly influenced the results obtained from the analyses.

9. Locating differences between means in both analyses through the use of post hoc tests (LSD) proved, on the whole, unsuccessful except for the G.P. group.

10. Endogenous patients responded as well to CBT as they did to drugs or combination treatment.

11. The three treatments resulted in different patterns of response for level of self-reported depression and hopelessness in subgroups of responders.

12. Duration of illness was the most important predictor of response to cognitive therapy, with or without drugs.

E. SUMMARY OF THE FINDINGS RELATIVE TO THE SPECIFIC HYPOTHESES TESTED

For the sake of simplicity and parsimony, the hypotheses associated with outcome will be discussed in relation to change scores only.

Hypothesis 1: that the hospital outpatients will have higher levels of depression than general practice patients is not supported: hospital and general practice patients had similar levels of self-reported and observed depression (p. 259).

Hypothesis 2: that the hospital outpatients will report more depressive symptoms than general practice patients is only supported in part: only two symptoms differentiated the hospital outpatients from the general practice patients (p. 254).

Hypothesis 3: that hospital outpatients will be more severely disturbed in terms of overall psychiatric symptoms than general practice patients is fully supported: the hospital patients had significantly higher total scores on the PSE (p. 259).

Hypothesis 4: that cognitive therapy will be superior to pharmacotherapy in terms of reducing the number of drop-outs from treatment is not supported.

There were no significant differences in the number of drop-outs between the two treatment groups in both populations (pp 261-262).

Hypothesis 5: that combination treatment will be superior to cognitive therapy and pharmacotherapy in reducing the number of drop-outs from treatment is not supported.

There were no significant differences in the number of drop-outs between the combination group and each of the other treatments in both populations (pp 261-262).

Hypothesis 6: that patients in all three treatment groups will have lower scores at the end of treatment than they did at the start of treatment on all mood measures is only supported in part for each population.

In the hospital outpatient sample, patients in all three treatment groups improved significantly on six out of seven mood measures (pp 283-284).

In the general practice sample, patients who received combination treatment and cognitive therapy alone improved significantly on all of the mood measures in the analysis of change scores (p. 283). However, patients in the pharmacotherapy group improved significantly on only three out of the seven mood measures (pp 283-284).

Hypothesis 7: that patients in all three treatment groups will have lower scores on hopelessness and higher scores on views of self, world, and future at the end of treatment than they did initially is fully supported for the hospital sample; for the general practice sample, the hypothesis receives only partial support.

In the hospital group all three treatments resulted in significantly lower scores on hopelessness and significantly higher scores on views of self, world and future (pp 283-284).

For the general practice group, combination treatment and cognitive therapy alone resulted in significantly lower scores on hopelessness and significantly higher scores on views of self, world and future. However, pharmacotherapy did not significantly reduce hopelessness. It did result

in significantly higher scores on views of the self and the world, but failed to produce significantly higher scores on view of the future (pp 283-284).

Hypothesis 8: that patients in all three treatment groups will perform better at the end of therapy than they did at the start on both behavioural tasks receives minimal support for each population.

In the hospital sample all three treatments failed to produce a significant increase in speech rate (counting speed); the three treatments did, however, produce significant increases in writing speed (p. 284).

For the general practice group, similar results were obtained except that pharmacotherapy failed to produce a significant increase in writing speed (p. 284).

Hypothesis 9: that patients assigned to the cognitive therapy group will show greater reductions in mood disturbance on all affective measures at the end of treatment compared to those assigned to pharmacotherapy is not supported for the hospital sample, but receives partial support for the general practice group.

In the hospital sample, cognitive therapy was only as effective as pharmacotherapy in reducing affective symptoms on five of seven mood measures. For the general practice group, however, cognitive therapy was significantly more effective than pharmacotherapy in reducing affective symptoms on three of seven mood measures (pp 288, 289, 290).

Hypothesis 10: that patients receiving cognitive therapy will show a greater reduction in hopelessness and higher scores on views of the self, the world, and the future than patients who received pharmacotherapy is not supported in the hospital group and receives partial support in the

general practice sample.

In the hospital group, cognitive therapy was not more effective than pharmacotherapy in decreasing hopelessness and in increasing scores on views of the self, the world and the future. For the general practice group, cognitive therapy was not more effective than drugs in reducing hopelessness or in increasing scores on views of the world and the future. However, cognitive therapy was significantly more effective than pharmacotherapy in increasing scores on view of the self, i.e. enhancing self-esteem (p. 290).

Hypothesis 11: that those who receive cognitive therapy will also perform better on the two behavioural tasks at the end of treatment compared to the pharmacotherapy group is not supported.

There were no significant differences between the two treatments in terms of increasing speech rate or writing speed (p. 286).

Hypothesis 12: that patients assigned to the combination of pharmacotherapy and cognitive therapy will show the greatest reduction in affective symptoms at the end of treatment on all mood measures is supported in part in the hospital population but is not supported in the general practice group.

In the hospital group, combination treatment resulted in the greatest overall reduction in affective symptoms compared to the other two treatment groups on five of seven mood measures (p. 288). Although the overall difference between combination treatment and the other treatments was statistically significant for self-reported depression (BDI) (pp 287, 290), the differences between combination treatment and cognitive therapy and combination therapy and drug therapy were not large enough to be statistically significant at the 5% level (p. 287).

For the general practice group, combination treatment was not more effective than cognitive therapy alone but it was significantly more effective than pharmacotherapy alone in reducing affective symptoms on three of seven mood measures (p. 290).

Hypothesis 13: that patients assigned to combination treatment will show the greatest reduction in hopelessness and the greatest increase in scores on views of the self, the world, and the future compared to the other treatment groups is supported only in part in the hospital population but not in the general practice group.

In the hospital sample, combination treatment was not more effective than either cognitive therapy or drugs in decreasing hopelessness. However, combination therapy resulted in the greatest increase in scores on views of the self, the world, and the future relative to the other two treatment groups (p. 288). When individual comparisons were made between combination treatment and cognitive therapy and combination treatment and drugs, no statistically significant differences emerged.

For the general practice group, combination treatment was not more effective than cognitive therapy alone but it was significantly more effective than pharmacotherapy alone in increasing scores on view of the self, i.e. enhancing self-esteem (p. 290).

Hypothesis 14: that combination therapy will result in better performance on the two behavioural measures at the end of treatment compared to each of the treatments on their own is not supported.

In both populations no significant differences emerged between the three treatments in terms of increasing speech rate or writing speed.

Hypothesis 15: that patients who respond to combination treatment will show a more rapid reduction in self-reported depression (BDI) than those who respond to cognitive therapy alone and pharmacotherapy alone is supported in part.

Responders to combination treatment showed a significantly more rapid reduction in depressed mood than responders in the pharmacotherapy group (p. 307). However, responders to combination treatment did not show a significantly more rapid reduction in depressed mood than responders to cognitive therapy alone (p. 307).

Hypothesis 16: that patients who respond to cognitive therapy and the combination of drugs and cognitive therapy will show a more rapid reduction in degree of hopelessness as measured on the hopelessness scale (HS) than those who respond to pharmacotherapy is fully supported.

Responders to cognitive therapy and combination treatment showed a significantly more rapid reduction in hopelessness than responders to drug therapy (P. 311).

CHAPTER SEVEN

DISCUSSION

A. ARE THERE DIFFERENCES BETWEEN THE TWO POPULATIONS SELECTED FOR INCLUSION IN THE STUDY?

In view of the wide range of patients selected for the trial and the increased interest in the treatment of psychiatric disturbances at the primary care level (Clare, 1978), it is clearly important to establish whether or not the psychiatric syndromes treated in community settings resemble those encountered in hospitals, since any consideration of the use of biochemical or psychological interventions in general practice must acknowledge the possibility that identical terms may be used to describe states that are in fact quite different.

Of the psychiatric syndromes encountered in general practice, depression is said to be one of the most common (e.g. Popoff, 1969; Goldberg and Kenel, 1975). It is, therefore, important to ask in what ways, if any, the depressive symptoms presented by general practice patients from the Leith surgery differ from those seen in patients who were screened at the Royal Edinburgh outpatient department.

It was expected that differences would be found between settings in that hospital patients would be more likely to present depressive symptoms which constituted an 'endogenous' pattern and would also manifest a significantly greater degree of depression than community-based patients (Wing et al., 1978; Shepherd et al., 1966).

Response to the PSE items used to meet the RDC for primary major depression indicated that depressed general practice patients and hospital patients report similar patterns of affective disturbance (also see appendix XI). Only two symptoms differentiated the samples, namely loss of libido and subjective anergia, with hospital patients reporting these significantly more often than general practice patients. Thus, one 'endogenous' symptom (loss of libido) did, in fact, distinguish the hospital patients from those seen in general practice.

Patients from the two clinics were also very similar in terms of intensity of depressed mood. There were no significant differences between the groups in self-reported (BDI) and observer-rated (HRS) depression. Mean scores for self-reported depression for both populations indicated a moderate degree of severity according to the British norms for the Beck Depression Inventory (Metcalfe and Goldman, 1965).

While patients from each referral source were similar in terms of symptom pattern and depth of depression, they were different from one another in several important respects. With regard to severity of illness, compared to general practice patients, the hospital patients were rated significantly higher on general psychiatric disturbance as assessed by total score on the PSE and had significantly lower self-esteem (AM, semantic differential). Moreover, they were significantly more chronic than the general practice patients who, in turn, were from significantly lower social class and were less educated than the hospital group.

It is interesting to compare these results with those reported in other studies (e.g. Blashki, 1972; Pilowsky et al., 1978). For instance, Pilowsky et al. (1978) found that depressed patients from inpatient and general practice settings reported the same degree of depressive severity

but showed different symptom patterns as measured by a depression questionnaire. Twice as many hospital patients were classified as 'non-endogenous' or 'endogenous' depreseed compared to the general practice patients, most of whom were assigned to a 'non-depressed' category.

Since no attempt was made to select patients according to specific diagnostic criteria, it is likely that the findings reported by Pilowsky et al. reflect the conceptual biases and diagnostic practices of the physicians in the two settings. Moreover, different patterns of affective disturbance might well be expected in comparisons of inpatient and community-based patients in that inpatients represent a small and highly selected subgroup of depressively ill people who tend to present with severe, psychotic, suicidal, therapy resistant and chronic depressions (Copeland et al., 1975; Helmchen, 1979).

In light of the findings of the present investigation, it appears possible that the measure used by Pilowsky et al. (1978) failed to discriminate depression in general practice patients because of problems related to the insensitivity of self-report scales (Hamilton, 1974, 1979, see chapter 5). Had a comprehensive and structured observer-rated instrument been used to gather the data, the difference in symptom pattern between patients from the two settings may not have been so marked.

The implications of not distinguishing between depressive subtypes can be readily appreciated, particularly as it has frequently been observed that depressions of the 'endogenous' type are more likely to respond to tricyclic antidepressants (Ball and Kiloh, 1959; Raskin et al., 1970; Paykel, 1972; Deykin and Demascio, 1972). However, the findings reported here challenge the assumption that these subtypes and intensity of depressed mood may be predicted on the basis of treatment setting.

The hospital group are a sicker population than the general practice patients in terms of overall level of psychiatric disturbance, though both groups obtained mean PSE total scores (Table, p.259) consistent with those reported by Wing et al. (1978) for inpatient and outpatient samples combined (i.e. PSE total > 21). This aspect of severity is mirrored by the finding that the hospital group had lower self-esteem and longer duration of illness than the general practice group.

The difference found between the groups on the cognitive measure suggests that low self-esteem (negative view of the self), a key component in Beck's cognitive triad, becomes more salient with increasing symptom severity. This would be predicted from the hierarchical nature of psychiatric diagnosis (Foulds, 1976). Various investigators have asked whether or not low self-esteem is preponderant at all levels and in all types of depression (e.g. Akiskal and McKinney, 1975; Blaney, 1977; Becker, 1977, 1979).

Another point may be raised concerning the two populations selected for inclusion in the study. Wing et al. (1978) note that in patients with depressed mood categorical differences in disorders may exist when there is an appearance of specific depressive symptoms with increased PSE total score, particularly psychotic ones (e.g. delusions, hallucinations). Although none of the patients in the study had psychotic symptoms, the hospital patients reported more specific symptoms (loss of libido, subjective anergia) along with higher PSE scores. Moreover, they were more chronic, the mean duration of illness being 91.4 weeks, or nearly two years. Therefore, it is possible that the hospital outpatient sample represents a subgroup of depressives within the 'umbrella' diagnosis of primary major depression. The differential response to treatment of this population is a critical issue and will be taken up later in the chapter.

With regard to demographic factors, the hospital patients were slightly older (43.7 ± 11.2) than the general practice group (39.9 ± 9.6) but the difference was not significant. Traditionally, depression has been viewed as a disorder of middle and later life. The data from this study, however, are consistent with recent reports of samples of treated patients which show younger ages between 30 and 40 (Becker, 1977; Paykel et al., 1979). One explanation for the relatively young age of patients selected for the trial lies in the younger age of neurotic compared to psychotic depressed patients, observed in investigations of depressive classification. For example, a study by Grad de Alarcon et al. (1975, in Paykel et al., 1979) showed the peak age for psychotic depression was in the 50's and that for neurotic depression in the 30's. Over the years milder illnesses have, in fact, featured more prominently in studies of treated depression with a notable shift towards younger women presenting with neurotic as opposed to psychotic depression (e.g. Rosenthal, 1966). This may reflect attenuation of illness by early treatment in the community; a change in referral patterns over time to include patients who previously might not have been regarded as ill or have accepted psychiatric treatment.

Many studies (see Weissman and Klerman, 1977; Silverman, 1968; Lehmann, 1971) show higher rates of depression in females in ratios from 2 : 1 to 3 : 1 and the results reported here are in line with these findings. In both populations women outnumbered men nearly 3 : 1 with a slightly higher proportion of men appearing in the hospital outpatient sample (36%) compared to general practice (26%).

Various explanations, biological and social, have been put forward for the preponderance of women in studies of depression (Weissman and Klerman, 1977). Biological theories centre around sex-linked genetic transmission (Gershon et al., 1971), hormonal effects as reflected in

premenstrual tension (Neu et al., 1974; Shader et al., 1970), post-partum depression (Paffenberger and McCabe, 1966; Pugh et al., 1963), the menopause (McKinley and Jeffries, 1974) and the use of oral contraceptives (Weissman and Slaby, 1973).

Psychosocial interpretations suggest that women differ from men in treatment-seeking behaviour (Weissman and Klerman, 1977) and in their preparedness to talk about their emotions. In many cultures crying and an appearance of helplessness are frequently viewed as feminine behaviours, or at least are more tolerable in women. Paykel and Rown (1979) note that other forms of deviance, most notably alcoholism, delinquency and adult crime, are more common in males.

Alternative hypotheses have been proposed specifying the pathways whereby women's disadvantaged status might contribute to clinical depression. Attention has been given, for example, to differential rates of mental illness among married and unmarried women. In a review of the area Gove (1972) found that rates of mental disorder were higher in married women than in married men, but for the single, divorced or widowed, findings were equivocal, with a tendency to higher rates in men. He proposed that, although marriage seems to have a protective effect for men, it may have a detrimental effect on women in that women are more likely to have to cope with stresses such as a low status role as housewife, several children, and financial dependence on the spouse, among others. The work of Brown and Harris (1978, see chapter 3) reflects the growing interest in this field; they observed high rates of affective disorders among women with young children.

The question of sex differences in the incidence of depression is complex and a full explanation probably involves incorporating several of

the theories mentioned above. It is particularly relevant to outcome studies such as the one reported here in light of findings which suggest a differential effect for treatment in women (e.g. Hordern et al., 1963; Hordern et al., 1965; MRC Trial, 1965).

The observed differences between the two populations in terms of education and social class are due primarily to selection factors. Nearly all of the hospital outpatients were referred by consultant psychiatrists connected with the Brain Metabolism Unit, Thomas Clouston Clinic which covers a large section of Southwest Edinburgh, a predominently middle class district of the City. It was anticipated that people referred from this catchment area would be 'typical' of patients who might be chosen for psychotherapy and yet, markedly different from those referred in Leith, most of whom were, in fact, less educated and fromlower social class. Another and more important reason for choosing these referral sources was to attract a broad range of patients for the trial. To date, investigations of the effectiveness of CBT have been limited to highly selected samples. For instance, Shaw (1977) and Taylor and Marshall (1977) both used university students; and Rush et al. (1977) had patients attending a university clinic and with an average of 14 years' education, as did Fuchs and Rehm (1977) in their study of self-control therapy. All of these subjects, therefore, tended to be of quite high verbal ability (Whitehead, 1979). These investigations do not allow conclusions as regards the possible relationship between treatment success and education or socio-economic level.

The results of this study, however, (table, p. 185) show that 50% of all patients admitted to the trial had minimal education (i.e. until the lowest possible school leaving age) and 27% came from lower social class. Within the general practice group 48% of the patients came from lower

socioeconomic levels (social classes IV and V).

Thus, the population selected for this project makes it possible to test the applicability of CBT to a wide variety of patients. It permits comment on whether or not low education patients can use the cognitive therapy style and whether or not the problems presented by patients of lower socioeconomic class can be dealt with in this way (Whitehead, 1979).

It is worth emphasising, in summary, that symptom pattern and moderately high total scores on the PSE indicated substantial psychopathology in both groups of patients.

B. ARE THERE DIFFERENCES BETWEEN PATIENTS WHO DROPPED OUT OF TREATMENT AND THOSE WHO COMPLETED THE TRIAL?

The attrition rate for both populations combined was 27 per cent, a finding consistent with figures reported for drug conditions in many controlled trials (Klerman et al., 1974; Weissman et al., 1974; Lasky, 1962; Weissman et al., 1979; Herceg-Baron et al., 1979), but considerably lower than those found in some psychotherapy studies (e.g. Daneman, 1961, 49%; Covi et al., 1974, 47%). More general practice patients dropped out of treatment (38%) compared to the hospital population (18%) but the difference was not significant. Furthermore, there were no differences in the attrition rates across treatments in each population.

Differential attrition poses a major threat to the internal validity of a treatment study, i.e. it influences the correctness with which observed effects can be attributed to the treatments being tested (Hollon and Beck, 1978; Lasky, 1962). However, since attrition was balanced across the treatments in each sample in this study, it need not be given much weight in the interpretation of outcome results. It is difficult to assess the

reasons for discontinuing treatment in the general practice but for the hospital patients, as contact was not lost, it is known that two were admitted to hospital, one woman's husband thought that she should be on drugs, two in cognitive therapy said it made them feel worse, one (duration of illness nine years) stopped coming after 10 weeks, two emigrated and one refused more treatment.

Although the specific aims of the present study did not include any plans to elicit directly the reasons why patients dropped out of treatment (e.g. spontaneous remission, treatment failures, or excessive side effects), an attempt was made to see how the rather large subgroup of noncompleters (both samples combined) differed at baseline from those who stayed in treatment.

The findings of the comparison of non-completers and completers for all variables examined at screening indicated that patients who dropped out of treatment reported greater intensity of depression (self-reported and observed), higher levels of inward and general irritability, and a more negative view of the environment. Moreover, noncompleters were also less educated than those who completed treatment.

The data suggest that the exact nature of the sample completing therapy might have been somewhat different from those accepted into the study. This places some limitation on the populations and situations to which the observed effects can legitimately be generalised. Even so, the results are interesting in that they raise questions concerning the validity of the diagnostic criteria used in this study (Spitzer et al., 1978) and more importantly perhaps, the problem of untreated depression in the community (Brown and Harris, 1978).

Given that most of the drop outs came from the general practice

setting, i.e. 15 of 24, or 63%, one explanation for the results might be that, while drop outs scored high on several severity measures of depression, they may have been suffering from some affective disturbance other than clinical depression when admitted into the trial. To put it another way, either the diagnostic criteria itself, or the way symptoms were elicited by the interviewer, resulted in a high proportion of 'false positive' diagnoses. In light of the findings cited earlier (chapter 5, p. 223) as regards the overinclusiveness of the RDC, it is possible that patients who experienced transient, yet acute, dysthymic states (e.g. anxiety, irritability, or the 'blues') following interpersonal or situational crises could have been incorrectly classified as depressed and admitted into the trial. As their condition improved from day to day, these patients might have decided that it was no longer necessary to consult a physician or remain in treatment. On the other hand, great care was taken to exclude patients reporting symptoms which suggested a diagnosis of primary anxiety (chapter 5, p.181) and this is likely to have minimised the admission of people presenting with general dysthymic discomfort or transient situational disturbances. Although some misclassification is inevitable whether either clinical or standardised diagnostic criteria are used to select patients, the findings reported here point to another explanation for the high proportion of drop outs from the Leith practice.

Brown and Harris (1978) argue that depression may be a consequence of aspects of social disadvantage. Whitehead (1979) takes up this argument, suggesting that whereas a well-off individual might become depressed because of his unrealistic appraisal of events, the poorer person might be quite accurate in his perception of environmental disadvantage. Presuming that the drop outs, many of whom came from lower social class, were 'real

depressives', it is conceivable that due to their significantly more negative appraisal of the world, these individuals decided that medical care on its own was unlikely to result in a meaningful change in their social circumstances. For some patients who discontinued treatment, depression may have developed, or at least may have been maintained by, an all-too-realistic appraisal of a deprived environment. In this context, it is notable that in addition to being more depressed than completers, the dropouts were significantly more inwardly irritable which suggests that those who have a negative, but possibly realistic, view of their environment, may blame themselves for their disadvantaged status. Abramson et al. (1978) have argued for the use of reattribution techniques in conjunction with 'environmental engineering' (e.g. rehousing families) as a means of reducing depression in such populations.

The fact that noncompleters were less educated than completers may be due to the higher number of general practice patients in the subgroup of dropouts. However, it is possible that less educated patients dropped out of the study because of failure to grasp the reasons why they were being treated or because of an inability to understand the principles and procedures of cognitive therapy.

Whatever the reasons for patients dropping out of treatment, the results of this study indicate that a considerable number of moderately depressed outpatients did not accept the professional help made available to them. While it is conceivable that some may not have been clinically depressed to begin with, there is little doubt that others went back to their homes having to cope with substantial psychopathology as well as routine 'problems in living'. True, various social support systems could have been available to help them deal with day to day stresses. However, if the patients

selected from the Leith area are in any way comprable to those found in Brown and Harris's survey in Camberwell, then in all likelihood these individuals returned to the community only to confront the social vacuum and loneliness which, according to Brown and Harris, feature so conspicuously in the lives of environmentally disadvantaged groups. The fact that over twice as many general practice patients had experienced loss due to separation, divorce and death relative to the hospital population lends weight to this argument (chapter 5, p. 185).

In summary, any investigator who conducts a planned treatment evaluation would like to be able to analyse data from all cases admitted to a trial. Yet in practice this is extremely difficult to accomplish; subjects drop out of a study during its course for many reasons. Unless an attempt is made to understand and deal with the effects of sample attrition, findings can be obscured, vitiated or biased.

This section considered differential attrition in the two clinical populations and across treatments within each subgroup. Differences between patients who completed treatment and those who did not were presented and possible reasons for premature termination of treatment put forward.

C. THE FINAL SAMPLE: ARE THERE DIFFERENCES BETWEEN PATIENTS FROM THE TWO SOURCES OF REFERRAL?

The data from the comparison of completers only across the two clinical populations indicate that the differences between the hospital and general practice samples were essentially similar to those found in the analysis of all patients admitted into the trial. That is, compared to the Leith sample, hospital outpatients were from higher social class, were better educated , had experienced more depressive episodes, and longer duration of illness, had significantly higher PSE total scores on average and lower

self-esteem. However, exclusion of the dropouts from both populations resulted in two other differences between the groups. The hospital completers were more hopeless and less outwardly irritable than the general practice completers, indicating that the samples which eventually completed therapy were, in fact, somewhat different from those screened into the study, with the hospital group being especially pessimistic about the future.

One explanation for this finding is that less hopeless hospital patients could have dropped out of therapy, which would have had the effect of raising the mean hopelessness score of the hospital completers relative to the general practice group. Bias on the part of the therapists who administered the cognitive treatment might account for relatively more hopeless people staying in the trial. The therapists' awareness of the reported connection between hopelessness and suicidal intent (Minkoff et al., 1973; Wetzel, 1976), plus the fact that they had been particularly concerned with mastering the techniques for identifying and reducing suicidal risk (Beck et al., 1979) could have resulted in their selectively attending to patients who initially verbalised strong feelings of hopelessness. On purely clinical grounds, this might be regarded as a trial success in that people most at risk to committing suicide were motivated to maintain contact with professional helpers.

With regard to the community-based sample, the mean hopelessness score in the general practice group could have been reduced if a relatively large minority of hopeless patients discontinued treatment. Presuming that a similar therapist bias occurred for the Leith patients, those who reported feelings of hopelessness would have received special attention and would, therefore, have been more likely to stay in treatment. Why is it, then, that a small number of hopeless patients from Leith may have

decided not to continue therapy? This is a difficult question and one which relates back to the problem of delineating the reasons for differential attrition in treatment studies.

However, Brown (1979) and Brown and Harris (1978) speculate that specific feelings of hopelessness are likely to occur as a consequence of exposure to stressful life events in the context of social deprivation. If this develops into a general feeling of hopelessness, it may form the central feature of a depressive disorder. Moreover, the chances of developing such a pessimistic attitude towards life may be increased considerably if, as Beck (1967, 1976) suggests, an individual has a learning history which did not foster the development and maintenance of a positive self concept. Given ongoing low self-esteem, a particular series of events or difficulties and a deprived environment, it is questionable whether the most competent therapist ('cognitive' or otherwise) would be able to help some individuals to adopt an optimistic view about controlling their circumstances. This clinical/social picture may apply to some Leith patients who were hopeless and discontinued treatment. For these people it is conceivable that therapist attention towards, and treatment of, hopelessness may not have been sufficient to motivate them to remain in treatment, let alone change their negative expectations about the future.

It is recognised that this is a speculative position and goes beyond the results presented in this thesis. Nevertheless, the discussion is relevant to sample attrition and again draws attention to the possible need for a broader, partly preventative approach, that might be best implemented outside the medical sphere (Brown and Harris, 1978; Brown, 1979; Abramson et al., 1978).

Patients' expectations about the nature of, and appropriate role

behaviours for, psycbotherapy significantly influence the course of treatment along a number of dimensions including duration (Garfield and Wolpin, 1963; Lorr and McNair, 1964), outcome (Lennard and Bernstein, 1960), patient discomfort (Baum and Felzer, 1964), involvement in the therapeutic process (Kamin and Caughlin, 1963; Levitt, 1966; White et al., 1964) and attrition rates (Overall and Aronson, 1963). It is likely that the differences between the two samples in terms of education and social class influenced expectations about cognitive therapy. This might help to explain not only the higher attrition in general practice, but why more hopeless hospital patients decided to complete therapy. In addition, the manner and level of enthusiasm with which the cognitive therapy project was described to the patients by doctors from the respective clinics would have affected treatment expectations significantly.

Before going on to discuss the outcome results, it is important to consider whether or not the completers from the hospital and general practice samples differed in terms of affective pattern, since some have argued that this has implications for response to treatment (e.g. Ruskin et al., 1976; Paykel, 1972). Using Spitzer's (Spitzer et al., 1978) criteria for endogenous depression, completers from the two clinics were classified as endogenous or non-endogenous depressed patients on the basis of their basal Hamilton Rating scores. There were no significant differences between the number of endogenous and non-endogenous patients in the two clinics, indicating that the hospital and general practice completers presented with essentially similar symptom patterns (see appendix XVIII). These data support the finding cited earlier (p. 328) with respect to the comparison of the groups on PSE symptoms and underscore the importance of avoiding assumptions about the specificity of depressive subtypes in relation to

treatment settings.

Summary

The first three sections of the discussion have been concerned with the sample of patients selected for the study. There is evidence that both populations experienced substantial psychopathology, that the hospital patients had more symptoms and were more severely disturbed than general practice patients, though both groups were similar in terms of level of depression, and that both populations had similar patterns of affective disturbance. Considerable attention was given to attrition, in particular how dropouts differed from completers. In some respects (e.g. intensity of depression, negative view of the environment) noncompleters were more severely disturbed than completers, a finding which must be weighted against conclusions concerning the applicability of the different treatments to depressed patients in general. Inferences were drawn from these data and possible reasons for dropping out of treatment were presented.

OUTCOME

D. This part of the discussion will focus on two questions:

1. Do hospital outpatients respond to each treatment modality?
2. Which treatment is most effective in alleviating symptoms in the hospital group?

In the hospital outpatient sample, pharmacotherapy (drug of choice), cognitive therapy, and a combination of the two were found to be effective in the treatment of outpatients diagnosed as having primary major depressive disorder. All three treatments resulted in substantial and statistically significant reduction in depressive symptomatology as documented by patient

self-reports and clinical evaluations. This was true, on the whole, for both absolute change and percentage change scores. Furthermore, all three treatmentswere associated with significant decreases in levels of self-reported anxiety and irritability. Since there was no placebo control group, it is possible that all patients could have improved spontaneously through the passage of time. This issue, however, will be taken up in more detail later.

Similarly, the three treatments produced significant changes in the expected directions along the cognitive parameters investigated, in particular those directly relevant to Beck's cognitive model of depression (views of the self, the world, and the future). That is, by the end of the treatment period, patients in each of the treatment groups reported higher self-esteem, a more positive view of the environment, and were more hopeful about the future. However, level of depression as measured by the behavioural tasks, by and large, did not change significantly over the course of treatment.

The efficacy of all three treatments was also supported by the finding that the number of responders and non-responders, as defined by decreased BDI score, did not differ significantly across the three treatments in the hospital group.

While all three treatments were successful in reducing the severity of affective symptoms and changing negative thoughts, analysis of covariance for change scores indicated that the combination of cognitive therapy and drugs produced the greatest improvement, followed by cognitive therapy and pharmacotherapy. This pattern held for seven of the eight variables which showed significant differential effects for treatment, i.e. anxiety (ANX), inward irritability (IN), general irritability (IRR), views of self

(AM), world (ENV) and future (FUT) and observed depression (HRS). Self-reported depression (BDI) did not show this pattern due to an interaction effect. Subsequent analysis of simple main effects for BDI change scores (controlling for duration of illness) resulted in a somewhat different pattern with combination treatment still showing the greatest significant reduction in depression, followed by pharmacotherapy and cognitive therapy. With regard to comparisons across the locations, hospital patients receiving combination therapy did significantly better than general practice patients who were assigned to the same treatment.

Significant differential effects for treatment in favour of combination therapy were also found when initial differences in levels of severity were taken into account in the analysis of covariance for percentage change scores. Combination treatment was superior to the other two treatments as documented by self-reported levels of anxiety (ANX), and self-esteem (AM) as well as clinical evaluations (HRS). There were minimal differences between the cognitive and drug therapy response rates. Again, an interaction effect was found for self-reported depression (BDI). After disentangling the interaction effects for BDI (covarying for duration of illness), a similar pattern to that found for change scores emerged in the present analysis. That is, the combination of cognitive therapy and drugs was more effective than either treatment on its own, followed by pharmacotherapy and cognitive therapy. In this instance, however, the difference among the treatments within the hospital population was not statistically significant. Thus the differential effects for treatment observed for BDI percentage change were due primarily to variance in an across-population comparison. In fact, one of the most striking results of this study was that, while both groups had almost identical mean percentage change scores, the hospital combination group did significantly better than the general practice

combination group. In this comparison, partialling out the effects of duration of illness had a particularly marked effect on outcome.

In the hospital group, post hoc tests to locate significant differences between means failed to discriminate between the treatment groups (except for the BDI change scores), a finding which could be explained by the very large variances (within group mean square error) observed in the different analyses.

When treatment adequacy is evaluated in terms of prevention of premature treatment termination, all three treatments were effective in keeping attrition very low, i.e. the number of dropouts was the same across the three treatments (COMB and DRUG = 3/13, 23%; CBT = 3/14, 21%). This is important since the data show that premature termination of treatment was associated with high levels of depressive symptomatology. Moreover, these figures compare favourably with those reported by Weissman et al. (1979) in their study of the efficacy of combined pharmacotherapy plus interpersonal psychotherapy. The lowest attrition rate for their hospital sample was 33% for combination treatment, compared with 52% for the psychotherapy alone group and 67% for pharmacotherapy alone.

The results of this study for the hospital group indicate that cognitive therapy works at least as well as pharmacotherapy and combining the two treatments has a partly additive effect. With respect to response in clinical research, a salient point is the difference between clinical meaningfulness and statistical difference (see Weissman, 1979; Whitehead, 1979; Hollon and Beck, 1978; Rush and Beck, 1978). Thus, for the hospital sample the clinical ranking of response is combination of cognitive therapy and drug of choice, cognitive therapy, and pharmacotherapy - the differences between each treatment being, on the whole, not statistically significant.

While the present study provides tentative support for the possible additive effect of combination treatment, the work of Rush and Watkins (in press) and Beck et al. (1979) indicates that pharmacotherapy may not enhance the effect of cognitive therapy administered on an individual basis. However, as seen in chapter four (p.148), methodological reasons for the discrepancy between these findings were anticipated. As regards the efficacy of cognitive therapy compared to other psychological treatments, Shaw (1977) and Morris (1975), for example, have shown that cognitive therapy is more effective than nondirective, behavioural, or insight oriented psychotherapy in the treatment of depressed patients.

The results of this study contrast with those reported by Rush et al. (1977) which showed the superiority of cognitive therapy over tricyclic pharmacotherapy. In the present investigation similar response rates were found for the two treatments. One reason for the discrepancy in outcome could be that in this study patients assigned to pharmacotherapy were given drugs suited to their individual needs (as judged by the clinicians) whereas in the Rush et al. study, one drug (imipramine) was prescribed for all patients. While imipramine is one of the most effective antidepressants available (Morris and Beck, 1974), Baldessarini (1977) has pointed out that from a clinical standpoint patients are likely to derive the most benefit from pharmacotherapy when they are put on a drug-of-choice regimen, since this provides the physician with maximum flexibility in the management of depressive symptoms.

In terms of methodological differences, another comparison might be made between the present study and that of Rush et al. Consistent with the literature relevant to the potential prophylactic effect of antidepressants (e.g. Klerman et al., 1974; Weissman et al., 1974), it was

decided that patients who showed a good response to drugs would be encouraged to take maintenance doses of medication up to six months after recovery. Thus, in the present study patients assigned to pharmacotherapy were still taking medication at the time of their final evaluation. However, in the Rush et al. study medication was tapered off during the last two weeks of the trial and discontinued before the patients had their last assessment. This could help to explain the observed superiority of cognitive therapy over pharmacotherapy in the Rush et al. study, particularly as it is likely that some patients in the cognitive therapy group were still involved in therapy immediately before the final evaluation. Although the results reported here show that cognitive therapy is, at best, only as effective as pharmacotherapy for hospital outpatients, it could be argued that the outcome is more striking insofar as it gives a realistic picture of the efficacy of cognitive therapy compared to the clinical practice of psychiatrists in a highly respected teaching hospital in Britain.

The question could also be raised as to whether the atmosphere and reputation of the University of Pennsylvania clinic, in which cognitive therapy was developed and refined, could account for cognitive therapy 'having the edge' over pharmacotherapy in the Rush et al. study. Treatment setting is an important ingredient in successful psychotherapy (Frank et al., 1978). The setting, because of its inherent therapeutic stimulus value, is a potent placebo variable. Direct comparisons of placebo response in different settings indicate that setting can induce placebo effects and affect treatment outcome in many ways (e.g. Rickels et al., 1966). Moreover, the reputation and milieu of a treatment research centre can influence the availability of resources for research as well as the amount

of dedication which members of staff bring to a project. In short, special research units such as the 'Center for Cognitive Therapy' cannot be said to be representative of the settings in which most depressed patients are likely to be treated.

In this context, in the Rush et al. study eighteen therapists participated in the treatment of 41 depressed outpatients, 19 of whom received cognitive therapy, making the therapist-to-patient ratio in the cognitive therapy group nearly 1 : 1. In addition, each therapist was systematically supervised every week by dedicated proponents of the treatment method (A.T. Beck, John Rush), a factor quite likely to have influenced the degree of motivation which the therapists brought into the treatment situation. By comparison, two self-trained (see p. 201) cognitive therapists administered treatment in the present investigation. They were supported by three psychiatrists and two general practitioners, none of whom had much investment in the theory and procedures of cognitive therapy. On the contrary, all were trained in and worked within a biological model of depression seen to be upheld by numerous drug studies (e.g. Morris and Beck, 1974). Bearing in mind that the 44 patients who received cognitive therapy, either with or without drugs, were seen by two therapists, the marked differences in resources and treatment settings and the potential impact these could have in terms of outcome are apparent.

It can be argued that this study represents the first successful 'field test' for cognitive therapy on the grounds that clinicians in a distant and independent institution, carrying a sizable caseload of depressed outpatients, obtained a response rate similar to that of pharmacotherapy. Moreover, the clinical psychologists involved in the study could hardly be regarded as part of an elite corps of cognitive therapists ('UK Vienna

Circle') since the trial in Edinburgh was begun before the recent upsurge of interest in Britain.

Finally, one other reason may be put forward to explain the less dramatic effect observed for cognitive therapy in this study relative to that reported by Rush et al. (1977). As duration of illness differentiated the hospital and general practice groups at a highly significant level, it was used as a covariate in the analyses of outcome for both change and percentage change scores. Thus in the present investigation degree of chronicity was statistically controlled. On the other hand, duration of illness was not controlled in the analysis reported by Rush et al., this in spite of what appeared to be a rather large difference in the distribution of chronic patients in the two treatment groups. To illustrate, 6 of 19 (31%) of their cognitive therapy group had been ill for over a year compared to 10 of 22 (45%) in the pharmacotherapy group. In light of findings which indicate that chronic depression is often associated with poor response to treatment (e.g. Rogers and Clay, 1975; Paykel, 1972, 1979; Freyhan, 1974), it is possible that the response rate in the pharmacotherapy group was somewhat inhibited due to the relatively high proportion of chronic depressives in that group. This would help to explain the superior effect observed for cognitive therapy over drugs in the Pennsylvania trial. Had these researchers used more sophisticated analyses, i.e. mean change scores corrected for degree of chronicity, they might have obtained results closer to those reported here.

The results of this study also contrast with a number of other studies which report the superiority of pharmacotherapy over various kinds of psychotherapy (Covi et al., 1974; Friedman, 1975). Potential methodological reasons for this descrepancy will be discussed shortly. However, from a clinical point of view, it has been suggested that the most parsimonious

explanation may relate to the nature of the psychological treatments used (Rush et al., 1977; Beck et al., 1979). It is conceivable that cognitive therapy is more successful than marital therapy (Friedman, 1975), social work counselling (Klerman et al., 1974), or traditional group therapy (Covi et al., 1974), since it was specifically developed for the treatment of depression. Extensive clinical and empirical work with depressed patients (see Beck, 1967, 1973, 1976) provide the framework for cognitive therapy which is specifically aimed at the central psychological problems of depression (e.g. negative views of self, world and future). Proponents of this treatment argue that the specificity and targeted approach of cognitive therapy may account for its success rate compared to those reported for other psychotherapies.

While this explanation for the effectiveness of cognitive therapy may well apply in relation to some other forms of psychotherapy, the results presented in this thesis suggest that pharmacotherapy alone can also modify the core psychological problems of depressive illness, as evidenced by the statistically significant changes observed along some cognitive parameters of the semantic differential. In the hospital group, pharmacotherapy resulted in patients adopting more positive attitudes about themselves and their environment as well as a hopeful outlook about personal future. Since pharmacotherapy and cognitive therapy showed similar levels of response on these parameters, it can be argued that both treatments have similar effects, i.e. changing negative cognitions, but achieve them through different modes of action. Furthermore, the combined approach with its partly additive effect and presumably multimodal action, may provide the best means of alleviating the psychological symptoms of depression.

The results of this study are in line with those reported by Weissman et al. (1979). They found that the combination of interpersonal psychotherapy (IPT) and pharmacotherapy was superior to IPT alone and pharmacotherapy alone and concluded that combination treatment has a partly additive effect in reducing the severity of symptoms in acutely depressed outpatients. However, in terms of the acceptability of psychotherapy to patients as measured by number of dropouts (e.g. Herceg-Brown et al., 1979), it seems that cognitive therapy, with or without drugs, is superior to IPT alone or in combination with drugs (see above, p. 345). Again, this may be due to the nature of the therapies employed. Cognitive therapy is a performance oriented treatment whereas IPT appears to be a verbally-based, self-explorative type of psychotherapy. In cognitive therapy depressed patients are encouraged to become active both inside and outside the treatment sessions and are taught to identify, control, and change negative thoughts associated with their depression, and to focus less on affective pain. It may be that non-behavioural forms of psychotherapy such as IPT, in which the message 'talk more and do less' is at least implied if not directly stated, sensitise the individual to affective discomfort, thereby making him more rather than less preoccupied with his symptoms. Not surprisingly in these circumstances, patients could be expected to discontinue treatment, thinking that psychotherapy has been ineffective.

The findings reported here indicate that cognitive therapy may hold great promise as a short term treatment for depressed hospital outpatients. It works as well as pharmacotherapy (drug of choice) and there is evidence that it can be more effective than tricyclic pharmacotherapy (Rush et al., 1977). However, combining cognitive therapy and pharmacotherapy is more effective than either treatment alone in terms of reducing depressive symptoms and changing negative thoughts.

E. This section of the discussion will address two questions:

1. Do general practice patients respond to each treatment modality?
2. Which treatment is most effective in alleviating symptoms in the general practice group?

In the general practice group the combination of drug of choice and short-term individual cognitive therapy and cognitive therapy alone were found to be effective in the treatment of depressed outpatients. Both treatments produced substantial and statistically significant reduction in depressive symptoms as measured by patient self-reports and clinical evaluations. This applied, in general, to absolute change and percentage change scores. Moreover, cognitive therapy, with or without drugs, was associated with significant decreases in levels of self-reported anxiety and irritability. On the other hand, statistically significant changes in the pharmacotherapy group were rare. This group, on the whole, demonstrated an unexpectedly poor response to therapy.

With regard to the cognitive measures, combination treatment and cognitive therapy alone resulted in significant changes in the expected direction, especially on those pertinent to cognitive theory (views of self, world and future). That is, patients who received these two forms of therapy reported more positive perceptions of themselves and their environment and were more hopeful about the future. However, pharmacotherapy did not produce significant changes in negative thoughts as measured by the semantic differential. Again, severity of depression as assessed by the behavioural tasks, by and large, did not change significantly over the course of treatment.

Cognitive therapy with or without drugs was successful in reducing the severity of affective symptoms and in changing negative thoughts. Analysis of covariance for change scores indicated that the combination of cognitive therapy and drugs and cognitive therapy alone resulted in similar levels of response on these parameters and that both treatments were more effective than pharmacotherapy alone. This pattern held for the eight variables which showed statistically significant differential effects for treatment, i.e. self-reported depression (BDI), anxiety, inward irritability, general irritability, views of self, world and future and observed depression.

Following up the interaction effects on the BDI with one-way analyses of covariance (covarying for chronicity) resulted in the same pattern, though the difference between treatments was not statistically significant. As mentioned earlier, a significant difference was found for combination treatment across locations with the hospital group showing greater improvement than the general practice group.

Statistically significant effects for treatment in favour of the combination and cognitive therapy groups were also observed in the analysis of covariance for percentage change scores. Combination treatment and cognitive therapy alone were superior to pharmacotherapy as measured by levels of self-reported depression, anxiety and self-esteem, as well as by clinical evaluation. Analyses of simple main effects for BDI within the general practice sample (covarying for chronicity) produced the same pattern, but once again, no significant differences emerged among the three treatments. As in the change score analyses, comparison across locations indicated the superiority of hospital combination treatment over combination treatment administered to general practice patients.

In the general practice group, post hoc tests for significant differences between means did, in fact, discriminate the treatment groups. For change scores, statistically significant differences emerged on three mood measures (anxiety, inward irritability, observer rating of depression) and one cognitive measure (view of self). The combination of cognitive therapy and drugs and cognitive therapy alone were significantly more effective than drugs but there were no significant differences between combination and cognitive therapy alone. For percentage change the only significant difference found was for observer rated depression where, again, cognitive therapy with or without drugs showed significantly greater reduction compared to the pharmacotherapy group. However, no significant differences emerged between the two cognitive therapy groups.

In terms of preventing drop-outs, all three treatments in general practice had high attrition rates, these being 38% (5/13) for combination treatment, 36% (5/14) for cognitive therapy and 42% (5/12) for drugs. Thus, no one treatment was much better than any other in minimising the number of drop-outs from therapy, against the expectation that cognitive therapy, particularly when used in conjunction with drugs (Herceg-Baron et al., 1979), would be more effective than drugs in preventing attrition. However, these findings could be attributed as much to the general problem of high attrition in general practice (Johnson, 1973) as to the idea that all of the treatments were relatively unacceptable to patients. High attrition in general practice is a matter of concern, since the data from this study shows that those who failed to complete treatment were more depressed than patients who completed therapy.

The results of the present investigation for the general practice group appear to indicate that cognitive therapy combined with drugs and

cognitive therapy alone are more effective than pharmacotherapy alone. In view of the minimal differences between the two cognitive therapy groups in terms of degree of response, there is no evidence to support the hypothesis that pharmacotherapy (drug-of-choice) enhances the effect of cognitive therapy administered on an individual basis to general practice patients (see Beck et al., 1979; Rush and Watkins).

The dramatic response to cognitive therapy and combination treatment and the poor response to drugs in general practice are by far the most striking findings in this outcome study. However, before drawing any conclusions about the efficacy of cognitive therapy compared to drug therapy in general practice, it is important to consider possible reasons for the extremely poor response in the pharmacotherapy group.

Several authors (e.g. Johnson, 1973; Wheatley, 1972) have commented on factors effecting poor compliance to drug regimens in general practice. Johnson (1973) implicated, in particular, the prescription of less than therapeutic doses, failure of supervision of the drug regimen, and poor contact with the doctor. For example, in Johnson's study, only 72% of the 73 patients investigated were given drugs in a dose that would be regarded as therapeutic. The potential benefit of therapy was considerably reduced by the patient's own actions, as 16% discontinued medication within a week, 41% within a two-week period, 59% within 21 days and after one month 68% of the patients had stopped taking their drugs. With regard to consultation rate, Johnson found that in the first six weeks of treatment just over a third (38%) of patients had had two consultations with the doctor and as few as 8% kept fortnightly appointments. In light of such findings, it is possible that the patients in the drug group in general practice failed to respond because of poor compliance.

In this study, though plasma levels were not monitored, all patients in the general practice were prescribed amitriptyline, or imipramine, at at least 100 - 150 mg daily. Because of the regular assessments, these patients had more supervision of drug regimen and more doctor contact than they would normally, seeing a hospital psychiatrist (Hamilton Rater) and a psychologist (who administered the self report questionnaires) every two weeks, in addition to seeing their G.P. for prescriptions. It can be argued, therefore, that the effect observed for pharmacotherapy is indeed genuine, suggesting that the sort of depression generally seen in a busy urban practice responds best to psychotherapy.

Putting aside the question of the specificity of pharmacotherapy in reducing depressive symptoms, perhaps a greater degree of response could have been expected from drugs on the basis of placebo effect alone. After all, large placebo response rates have been obtained in both anxiety neurosis and neurotic depression, this being in the order of 50% (e.g. Wheatley, 1968, 1969, 1970, 1972). Many factors related to patient and situational variables affect the degree of placebo response (see Shapiro, 1978, for an excellent review). However, one inescapable fact is that placebo effects are only as good as the attitudes the attending physician conveys about treatment.

Negative staff attitudes can decrease the efficacy of active medication (Sabshin and Ramot, 1956, in Shapiro, 1978). The effect of a placebo can be greatly diminished, from 70 to 25 per cent, if a negative attitude towards medication is communicated to a patient (Volgyesi, 1954). In another investigation, for example, patients treated with placebos showed greater improvement than those on tranquillisers or behaviour therapy, a finding the researchers attributed to the bias of nurses against pharmaco-

therapy and habit-training (Baker and Thorpe, 1956, in Shapiro, 1978).

In terms of predicting response to drugs in general practice, Wheatley (1972),Sheard (1963, 1964) point out that the more optimistic the attitude of doctor and patient, the more likely is an effective response to treatment. In this context, it should be noted that the doctors from the Leith practice, while sympathetic to their patient's problems and conscientious in the administration of pharmacotherapy, often expressed concern about the adequacy of this form of treatment, not so much because they doubted the potential efficacy of drugs in reducing depressive symptoms, but because in their eyes it offered little in the way of helping patients to deal effectively with overwhelming social problems (e.g. poor housing, family disharmony, financial difficulties). Thus, it is conceivable that the G.P.s, as Beck (1976) suggests sometimes happens to cognitive therapists, adopted the <u>negative cognitive set</u> expressed by their patients and inadvertently conveyed a negative attitude about the effectiveness of the drug treatment. This would have had all the more impact if, as is likely, the doctors believed implicitly that these depressions were reality-based and, as such, perhaps refractory to purely physical treatments. This would help to account for the very poor response in the general practice drug group.

Another explanation for the low level of response in the drug group could be that a disproportionately high number of treatment resistant patients had been assigned to the pharmacotherapy group. Wheatley (1972), in discussing predictors of response in general practice, has noted that response is better when there is no previous therapy, or when it has been previously successful. While degree of chronicity was controlled in the statistical analyses, history of previous treatments was

not. Even if this had been planned initially it would have been extremely difficult to identify 'treatment resistant' patients in the general practice, not only because of the difficulties mentioned above (e.g. poor compliance, low doses of tricyclics), but because of the imprecise manner in which patient records are sometimes kept in general practice. For instance, in the Leith practice it was not unusual for the hard-pressed G.P. to omit a note in the record indicating that a patient was depressed and that he had, in fact, been given a prescription for antidepressants.

Controlling for previous treatments is an important methodological issue and should be considered in the planning of future outcome studies in general practice.

With regard to combination therapy, the question could be raised as to whether good compliance with drug regimen, stimulated by regular contact with the cognitive therapist, could account for the dramatic effect observed in that group. Indeed, it is likely that compliance in the combination treatment was better than in the pharmacotherapy group, since the therapist stressed the importance of taking antidepressants at the start of treatment and, as needed, throughout the course of cognitive therapy. In fact, patients were encouraged to take their medication regularly as part of 'cognitive therapy homework'. That is, in conjunction with, for example, monitoring negative thoughts or increasing activities gradually, patients worked with the therapist in the implementation of the drug regimen. So, if the patient had been prescribed amitriptyline 100 mg to be taken each night, he would record this on his activity schedule as a task to be completed daily. Thus, it is possible that patients in the combination group improved as a result of pharmacotherapy alone, particularly

as drug schedules were fitted to patients daily routines (Haynes, 1976). However, the positive findings for cognitive therapy obtained by the present researchers and others (Rush et al., 1977; Rush and Watkins; Taylor and Marshall, 1977; Shaw, 1977) greatly weakens this argument.

An important aspect of this psychotherapy investigation is that it involved patients with minimal education, many of whom came from lower social classes. The results indicate that patients with only a basic education can, in fact, respond to cognitive therapy. It is worth noting that, on the basis of decreased BDI score (50% or $\leq$ 8), all eight completers in the cognitive therapy group and eight of nine completers in the combination group showed significant clinical improvement. In short, it seems as though a patient need not be a 'Rhodes Scholar' in order to benefit from cognitive therapy.

This contention, however, has to be expanded by considering the cognitive therapy approach as it was used specifically in the general practice setting. From the very beginning of this project it was recognised that conducting psychotherapy with a relatively disadvantaged population such as that found in Leith might present special problems. For instance, many studies indicate that psychotherapists working with lower social class patients are confronted with two major difficulties: a consistently high rate of premature defection from treatment and a poor response to conventional psychotherapies (Lorion , 1975, 1978). It was thought that both of these problems could reflect a single basic problem, namely poor adherence, either to the appointment schedule and/or to the procedures of treatment (Rush and Watkins, in press).

For lower social class people both pragmatic and attitudinal factors

contribute to early termination and/or poor adherence to treatment procedures. Occupational and child care problems as well as transportation are the most frequently encountered (Lorion, 1978). The lower class mother is often a single parent who has no reliable child care resources to allow her to seek treatment, let alone attend psychotherapy sessions regularly (Brown and Harris, 1978).

With regard to attitudinal problems, Lorion (1975, 1978) has pointed out that lower class patients (1) often do not regard psychotherapy as a treatment of choice, (2) are unsure about the relevance or effectiveness of psychotherapy, (3) are frightened by and ashamed of requiring therapy, (4) are perturbed that their problems may have an emotional basis, (5) may feel defensive and threatened by the prospect of self-evaluation and (6) may come to the therapist seeking direct advice and guidance for problems, rather than emotional support and specific treatment (Rush and Watkins, in press).

Premature termination and poor adherence in lower social class patients are also likely to be associated with therapist attitudes such as (1) problems in developing an understanding approach (empathy), (2) frustration at being unable to conduct therapy within a 'traditional therapeutic role' and (3) negative feelings towards and perhaps disapproval of the patient's behaviour (Lorion, 1975).

In light of this information, special attention was given to the 'socialisation stage' of treatment in which the therapist and patient dealt with one another's expectations about therapy. Considerable time was spent with general practice patients trying to align their feelings, attitudes and expectations with what they were likely to experience over the course of treatment. This preparatory approach finds support, for instance, in

an investigation by Heitler (1973). He found that patients from lower social class who were systematically prepared for group psychotherapy became more readily and adequately involved in the tasks of the treatment situation.

In the present study, attention to the problem of discrepancy of expectations sometimes involved as many as three sessions. From a clinical stand point, this appeared to reduce the amount of effort required to convey successfully the basic concepts and techniques of cognitive therapy. With adequate preparation it seemed as though patients were more prepared to become active in therapy, e.g. giving the therapist examples from their own experience of the connection between thoughts and emotions.

Another important aspect of administering cognitive therapy to the general practice patients was that the therapist consistently tried to avoid using 'psychological jargon'. Terms like 'automatic thoughts', 'operating from a false premise', 'agendas', etc. were promptly abandoned in favour of what seemed like more reality-based words or phrases, such as 'harsh, unfair criticisms of yourself', 'belief (thinking habits) which might cause you more distress than need be', 'plans for today's session' and so on. Throughout treatment the therapist used this common sense framework to facilitate the collaborative approach which Beck et al. (1979) regard as a hallmark feature of cognitive therapy.

It is notable that before discussing any of the technical aspects of cognitive therapy in their manual, Beck et al. (1979) emphasise the importance of using cognitive techniques within a psychotherapeutic atmosphere characterised by trust, warmth, empathy, etc. To this end the therapist regarded himself as a potential confidant to each patient

and as much as possible attempted to maximise the interaction of non-specific and specific factors in the therapy situation. Quite frequently this necessitated putting aside the 'technology' of cognitive treatment to listen to a patient and perhaps reflect with her about the pressures of, for example, living in overcrowded conditions (e.g. high noise levels, lack of privacy), or being employed as a domestic worker (some people held two jobs) and still having to care for the children and household. This is not to say, however, that 'empathic support' was used as a substitute for the cognitive techniques. In accordance with the cognitive therapy regimen, all of the sessions were highly structured, involving focused discussions relevant to the cognitive model of depression and homework assignments (e.g. monitoring negative thoughts, graded tasks).

In this context, patients in the general practice seemed to adhere best to the behavioural performance-based procedures. For instance, asking patients to do things systematically such as reading a book for 15 minutes a day, keeping a record of waking activities, gradually going out to socialise, cooking meals, etc. resulted in good compliance. On the other hand, while these patients were very capable of carrying on a cognitive therapy style dialogue and 'catching negative thoughts' within the sessions, it was very difficult to get them to record these thoughts and practise cognitive strategies in their home environment. Thus, for the cognitively oriented tasks, compliance was quite poor in the general practice group.

Cognitive therapy in the general practice setting also demanded flexibility and a willingness on the part of the therapist to depart from the routine employed with hospital patients. For example, child care difficulties often prevented women in Leith from attending twice a week and it became necessary for the therapist to comply with a once-a-week

format until such time as a twice weekly arrangement could be made. Rather than rigidly adhering to a designated appointment schedule it was necessary on occasions to employ 30 minute sessions in order to accommodate a patient's domestic and family responsibilities (e.g. children coming home from school early, having to get home to meet a husband who had unexpectedly changed his work schedule from night to day shift, or other domestic crises).

The foregoing section of the discussion has been concerned with the way that cognitive therapy was conducted in the Leith practice. It is recognised that most of the information discussed was anecdotal in nature and based primarily on observations made in the clinic over the course of the research project. However, in view of the unexpected finding that lower social class depressed patients respond well to cognitive therapy, a clinically-based elaboration of the results seemed entirely appropriate.

The positive findings in the Leith surgery have implications for the implementation of cognitive-behavioural techniques in general practice. The use of traditional methods of psychotherapy within the general practice setting are likely to be impractical. Even for the psychologically-oriented and highly motivated G.P., psychodynamic interpretations made in the hurried climate of a busy urban practice would probably be invalid, deterimental or too painful for the depressed patient to tolerate.

A number of adaptations to psychotherapy have been suggested as being appropriate for the general practice situation seen in Western Europe. For example, Balint (1957), Balint and Norell (1976, in Clare, 1978) have argued for a shift away from the conventional role of the doctor as a diagnostician and 'towards that of a listener able to 'tune in', follow the patient's lead, and allow the patient to make use of him' (Clare, 1978 p. 172). According to Clare, the notion of 'a flash' used to describe

the point when physician and patient simultaneously understand each other is an essential component of this approach. However, in light of the complex nature of clinical depression and the inherent risk of the patient committing suicide, not everyone would agree that this form of psychotherapy is likely to be helpful.

Other suggestions include the application to family practice of the theories of transectional analysis (Browne and Freeling, 1967) and the use of behavioural interventions (e.g. Marks, 1974, 1975). However, there is little evidence as yet that such techniques are permeating the main body of family practice (Clare, 1978).

The results of the present investigation indicate that cognitive therapy may be a promising technique for the short term treatment of depressed patients in family practice. Compared to traditional methods of psychotherapy, it is specifically designed to deal with the core psychological aspects of depression (e.g. hopelessness), is relatively quick in reducing depressive symptoms, taking up to 3 months on average, and can be applied to a wide range of patients, including those with a basic education. Furthermore, it may have the added advantage of giving the patient a set of coping strategies which he can use to prevent the onset of subsequent depressive episodes.

Whether cognitive therapy should be reserved for clinical psychologists and psychiatrists or should be extended to family doctors, social workers, community nurses and health visitors is an intriguing question which remains to be answered.

To conclude, combining drugs and psychotherapy in the general practice setting did not have a negative interaction but neither was there the partly additive effect seen in the hospital outpatient group. The inter-

action appears to have been reciprocal (Klerman, 1975; Hollon and Beck, 1978), i.e. the effect being similar to the better of the two individual treatments.

Outcome

F. This section will address the question: Do the three treatments show different effects in terms of the degree of response at various stages of therapy?

The results indicate that combination treatment, cognitive therapy, and pharmacotherapy result in different response patterns over the course of therapy. These were evident on the two measures examined, i.e. self-reported depression (BDI) and hopelessness (HS), in subgroups of responders to each treatment mode (hospital and general practice samples combined).

With regard to depth of depression (BDI), pharmacotherapy showed a stable, gradual effect in reducing depressed mood with the first significant improvement occurring at approximately six weeks into treatment. While cognitive therapy was quicker in significantly decreasing depressed mood (two weeks), it produced a rather unstable, erratic response pattern. However, combination treatment resulted in both a quick (i.e. statistically significant reductions at two weeks and again between two and four weeks) and stable reduction in depressed mood.

When the different levels of response of the three treatments were compared at consecutive occasions of testing, there were no significant differences between pharmacotherapy and cognitive therapy. However, combination therapy resulted in significantly greater improvement at six and eight weeks approximately, indicating that combining cognitive therapy and drugs produces the quickest reduction in intensity of depression.

The differential response rates of the three treatments were demonstrated more dramatically on the measurement of hopelessness. Pharmacotherapy showed a very delayed effect in decreasing hopelessness, i.e. significant improvement in the group of responders did not occur until approximately 10 weeks into treatment. In contrast, cognitive therapy produced a significant reduction in hopelessness at approximately two weeks into therapy. This rate of improvement was surpassed by combination treatment which not only resulted in significant improvement at two weeks but was followed by another significant reduction in hopelessness between weeks two and six approximately.

Comparing the three treatment groups with each other at consecutive occasions of testing showed that cognitive therapy, with or without drugs, was significantly quicker than drugs alone in decreasing hopelessness. It is notable that when cognitive therapy and combination treatment were compared at the different assessment times, no statistically significant differences emerged which suggests that there is an equivalent effect for the two treatments in reducing hopelessness.

These findings support the argument that cognitive therapy in combination with drugs may be one of the best ways to treat depressed outpatients. Looking at progress at various stages of therapy, it appears that combination treatment in particular, but cognitive therapy as well, have a sharper and quicker effect, especially on a core psychological symptom of depression, namely hopelessness. As level of hopelessness has been found to be a better predictor of suicide than depth of depression (Minkoff et al., 1973), this finding may indicate that cognitive therapy alone or in combination with drugs may be more effective than drugs alone in diminishing the risk of suicide in depressed patients.

These data take on added significance in view of the concern expressed by numerous researchers (e.g. Silverstone et al., 1974; Morris and Beck, 1974) that pharmacotherapy, in spite of the not inconsiderable success of the approach, often fails to bring quick relief to the patient. It takes time for drugs to work and even with the finest of drug regimens, the risk of depressed outpatients committing suicide remains high, particularly in the short term. By adding cognitive therapy to drugs, not only could it be possible to lower the risk of suicide, but perhaps through some interactive process (cognitive x neurochemical change), it could be possible to speed up the onset of drug effects.

Summary

The last three sections of the chapter (D, E, F) were directed towards a discussion of the relative effectiveness of combination treatment, cognitive therapy and pharmacotherapy (drug-of-choice) in the treatment of depressed hospital and general practice patients. For the hospital sample, there is evidence that cognitive therapy is at least as effective as drugs and that combining cognitive therapy and drugs has a partly additive effect. In general practice, both combination treatment and cognitive therapy alone were superior to drugs in reducing depressive symptoms. It was argued that this superior efficacy might have been due to poor compliance to drug regimen in the drug alone group. Whether this is so or the superiority of cognitive therapy is due to a specific effect, the advantage of giving cognitive therapy to people who choose not to take drugs is apparent.

Considerable attention was given to describing how cognitive therapy was adapted clinically to suit the special problems faced with general practice patients, most of whom were from lower social classes than the

hospital patients or patients treated with cognitive therapy in previous studies.

Finally, it was argued that combination treatment and cognitive therapy may give the patient quick relief in terms of reducing depressed mood and pessimism.

G. WHICH VARIABLES BEST PREDICT RESPONSE TO EACH TREATMENT MODALITY?

Using percentage changes on the BDI as the criterion variable, most of the outcome variables were employed as predictor variables in three multiple regression analyses, one for each treatment, to try and establish which variables would best predict response.

The data indicated that for cognitive therapy and drugs combined, long duration of illness was a negative predictor and high level of education a positive predictor. For cognitive therapy alone, again long duration of illness was a negative predictor as was a relatively positive view of the world; in other words, patients with a relatively high negative view of the world did better on cognitive therapy. With respect to drugs, being a patient attending a general practice such as the Leith surgery and having a large discrepancy between current self-image and concept of ideal self (low self-esteem) were negative predictors. To state these in positive terms: (1) the shorter the length of illness and the higher the level of education, the greater the improvement with combination treatment, (2) the shorter the length of illness and the more negative the expressed view of the world, the greater the improvement with cognitive therapy alone, and (3) being treated in a psychiatric outpatient department and reporting relatively high self-esteem will result in

greater improvement with drugs.

Thus, the prognosis for people receiving the different treatments was largely determined by factors dependent on the natural history of the depressive disorder, treatment setting, educational level and cognitive parameters.

The greater improvement predicted for patients who present with short duration of illness was an expected finding. Prior chronicity, as reflected by longer current depressive episode has been implicated as a poor predictor of response to pharmacotherapy (Kerr et al., 1972; Paykel et al., 1974). Poor outcome has also been associated with a history of more previous illnesses (Paykel et al., 1973; Wittenborn et al., 1973), this being perhaps another dimension of prior chronicity (Paykel, 1979). Kiloh et al. (1962) reported that insidious onset without precipitants but with duration less than one year indicated favourable response to chemotherapy; and in a recent study of outcome of antidepressant drug treatment in psychiatric outpatients and general practice, Tyrer et al. (1980) found that absence of chronic illness was significantly related to a good outcome.

That treatment setting predicted outcome in the pharmacotherapy group in this particular investigation is hardly surprising. The poor response to drugs in the general practice group, as seen in the analyses of covariance, was simply confirmed in the multiple regression analysis.

A number of empirical studies indicate a positive relationship between improvement and educational attainment, this being true, on the whole, for both pharmacotherapy (Raskin et al., 1974; Rickels et al., 1964; Downing and Rickels, 1972; Bielski and Friedel, 1976) and psychotherapy (Garfield, 1978; Luborsky et al., 1971). However, as Garfield points out, and as is so common in psychotherapy research in particular, the main

criterion of outcome in most of these investigations has been therapist ratings. These ratings could well have suffered from a lack of objectivity because of therapists' possible preference for middle-class patients. Meltzoff and Kornreich (1970), in their review of the area, reached the conclusion that the evidence was not entirely convincing and that educational level is not a potent factor in prognosis. It is to be noted that in this investigation educational level was not a predictor of response to cognitive therapy alone, the treatment which, in practice, was most obviously linked to the patient's ability to use the terms and concepts of the therapist.

As discussed above, recent work on preparing the patient for psychotherapy suggests that educational level, and perhaps intelligence, may be less significant than originally thought. It may be more important to provide the patient with specific information about the process of psychotherapy and his required participation than to attempt to establish the basic levels of education and intelligence needed (Bloch, 1979). This possibility is supported by the finding that the general practice patients were able to use the cognitive approach after <u>detailed discussion</u> of what they were likely to experience in the course of treatment.

The relationship between specific cognitive aspects of depression (e.g. components of the negative cognitive triad) and outcome has not received much attention in the literature. However, the findings reported here suggest that this could be a fruitful area of research. It may be that patients who are preoccupied with certain kinds of psychological difficulties (e.g. negative bias against the world and themselves) may benefit most from time-limited psychotherapies designed specifically to alleviate these problems and which could be given alone or in conjunction with drugs.

That cognitive factors were related to outcome as opposed to other symptoms (e.g. anxiety, depressed mood) point to the importance of attitudes (Beck, 1976) as potential predictors of outcome.

With regard to premorbid personality, a history of 'neurotic' traits has been reported to predict a poor response to tricyclic drugs (Kiloh et al., 1962; Deykin et al., 1972). By way of comparison, Kupfer et al. (1975), studying unipolar depressed outpatients, found premorbid traits of chronic anxiety and obsessiveness to be related with a positive response to tricyclics in patients with recurrent endogenous depression, who are said to have premorbid traits of excessive orderliness, conscientiousness and dependency (Von Zersson, 1977), it is clear that a history of at least certain 'neurotic' traits does not militate against response to drugs. Some of the confusion in this area may result from differing views about the nature of the term 'neurotic traits' (Stern et al., 1980).

These studies notwithstanding, relatively little attention has been paid in the research literature to the role of personality as a predictor of clinical course and long term outcome (Weissman et al., 1978). The research that has been done has involved the use of very broad personality factors such as neuroticism and extraversion (Eysenck et al., 1964). However, the measures used (e.g. the Maudsley Personality Inventory) do not assess dimensions of personality such as regulation of hostility, dependency and obsessional features that have been identified in the clinical literature as related to predisposition to depression. Furthermore, none of the measures are geared to assess specific schemas which Beck (1976) sees as crucial aetiological features of depression. In theory, it is schemas, or cognitive templates, that selectively filter

experience in specific ways which result in the affective temperaments mentioned above, and which make the individual prone to being highly self-critical, seeing the world as overwhelmingly demanding, and regarding his future in a pessimistic light.

Follow-up studies of depressed subjects should be conducted which include, in addition to broad personality measures, some of the newer measures developed specifically for depressed patients, e.g. Dysfunctional Attitudes Scale (Weissman, 1978, personal communication) and being tested by members of the University of Pennsylvania staff.

The study of the relationship between aspects of personality and long term outcome is an important area as evidenced by a recent article by Weissman et al. (1978). In a follow-up investigation of female depressed patients who had been treated on an outpatient basis, they found that the most important predictor of long-term outcome (i.e. eight months, two and four years after the acute episode) was personality as assessed by the neuroticism scale of the Maudsley Personality Inventory. Only the neuroticism scale differentiated chronic and non-chronic patients, with chronic patients being more neurotic than non-chronic patients. It may be that highly neurotic individuals have a repertoire of dysfunctional cognitive schemas which put them at greater risk for developing chronicity. Future longitudinal studies of depression should consider the possible relationship between dysfunctional schemas (attitudes), neuroticism and outcome.

In this study, multiple regression analysis was used to determine predictors of response to the combination of cognitive therapy and drugs, cognitive therapy alone and pharmacotherapy. It should be remembered

however, that some criticisms of regression techniques have been put forward which undermine the confidence that can be placed in the findings reported here. For example, Klein et al. (1969, in Paykel, 1979) have noted that psychiatric rating variables frequently do not meet the assumptions for regression. Paykel (1979) has summarised the problem as follows: 'Distributions are often skewed, and may be J-shaped or U-shaped. Relationships between variables may be heteroscedastic, curvilinear rather than linear, and interactive rather than additive. Findings based on multiple regression are often hard to replicate. In maximising the variance in the dependent variable accounted for by the predictor variables, the multivariate technique may capitalise heavily on relationships which are chance findings or are specific to the sample and cannot be replicated in new samples' (P. 195).

In spite of these difficulties, the results from the multiple regression provide some useful information in that they confirm the negative relationship between long duration of illness and short-term outcome reported by many researchers (see above, also Rogers and Clay, 1975, chapter 2, p. 16) and raise questions for future research. As an extension to the regression method, an attempt was made further to identify variables which could predict response to each treatment by computing a discriminant function analysis using hypothetical categories, i.e. predicted treatment groups comprising 'cases' assigned to each group on the basis of the regression equations obtained from the multiple regression analysis. In view of the highly abstract nature of this last analysis, the data based on it need not be included as a central feature of the discussion. However, a complete description of the rationale and the analysis itself appears in Appendix XIX.

A final point may be raised as regards prediction of response to each

of the three treatments investigated in this thesis. The results indicated that pattern of symptoms as defined by the categories 'endogenous' and 'non-endogenous' did not predict response to combination treatment, cognitive therapy alone, or pharmacotherapy alone. That is, endogenous patients responded as well to cognitive therapy as they did to drugs and combination treatment. To the extent that it is valid to speak in terms of 'non-psychotic endogenous depression', the data are consistent with earlier studies (e.g. Abraham et al., 1963; Spear et al., 1964) which failed to find a relationship between endogenous symptom pattern and specific response to chemotherapy. On the other hand, the results contrast sharply with studies which have shown a strong association between symptom pattern and response to pharmacotherapy (Ball and Kiloh, 1959; Kiloh et al., 1962). The disagreement among researchers in this area is due perhaps to the differing conceptions of what constitutes 'endogenous' depression. In fact, the criteria used to define 'endogenous' depression in this thesis, i.e. Spitzer's RDC (Spitzer et al., 1978), have been criticised for being overinclusive (see chapter 5, p. 222).

H. LIMITATIONS IN THE METHODOLOGY

Previous sections of the discussion have touched upon various interpretations of the findings. The next section will consider possible methodological flaws or other factors that could account for the results.

One flaw in the design of the present study is that no provisions were made to control for previous treatment response and spontaneous remission. It is possible that the good outcome seen for cognitive therapy alone in both populations could have been obtained because the patients would have got better no matter what kind of treatment they

received. This criticism may be addressed in two ways. First, the hospital group had a long history of depression and many had been treated without success before their admission into the study. Moreover, both groups reported moderate levels of depression at intake and considerable levels of psycopathology as documented by PSE ratings. Thus, for the hospital group at least, the psychiatric histories highlight a group more likely to be treatment resistant than responsive to therapy. Secondly, as in the Rush et al. (1977) study, the drug treatment group in the hospital sample showed a good response rate (77%) as defined by at least 50% decrease in self-reported depression (BDI). The degree of response exceeds the median rate for spontaneous remission (43%, range 18-67%) discussed by Bergin et al. (1978) in their review, as do the response rates for the other treatment groups, except for the general practice drug group, i.e. HOSPITAL SAMPLE: combination = 77%, cognitive therapy = 57%; GENERAL PRACTICE: combination = 99%, cognitive therapy = 100%, drugs = 14%. By way of comparison, only 50% of the patients in the pharmacotherapy group in the Rush et al. study met this criterion of significant clinical improvement (50% of pre-treatment BDI scores). The very fact that the general practice drug group showed such a poor response also argues against the idea that the patients would have responded to virtually any intervention.

Another criticism that can be made is that the doctors in both clinics initially selected patients for referral to the trial on the basis of individual implicit clinical criteria. For example, a hospital psychiatrist could have referred mainly therapy resistant cases, thinking that cognitive therapy was 'worth a try', whereas the general practitioner could have selected socially disadvantaged people who, he reckoned, might benefit from regular talks with 'a good listener' who could offer advice about the

best way to deal with social problems. Such differential selection is a serious problem which threatens the extent to which results can be generalised to a wider population.

While pre-selection may well have occurred in both clinics and was outwith the control of the experimenters, treatment assignment was random. No attempt was made to match the treatment groups for age, sex, history of illness, social class, educational level or severity of psychiatric symptoms (PSE). However, the dimensions which differentiated the two populations, i.e. duration of illness, PSE total scores, educational level and social class, were controlled in the statistical analyses, making the treatment groups relatively comparable.

A potentially serious flaw in the design is that there could have been considerable variation in the way that the clinical assessors rated patients on the Hamilton scale. Obtaining reliability data proved to be very difficult as a number of consultant psychiatrists and senior registrars conducted the assessments on a rotational basis. An attempt was made initially to have both an observer and an interviewer at each assessment which would have provided the necessary information to do a proper reliability check. However, time constraints and limited resources prohibited adequate implementation of this plan.

In this context, it could be argued that, since the assessors were not always blind as to treatment allocation, bias in favour of combination treatment, or cognitive therapy alone may have affected their evaluations. However, one reassuring point may be raised concerning the objectivity of the assessors. Since this study was carried out by the Brain Metabolism Unit, it is fair to say that the bias of the staff is strongly pharmacological, yet the ratings of the progress of patients on drugs do not reflect this

bias. Furthermore, it is doubtful that blindness can be maintained reliably in treatment studies comparing drugs with a psychotherapy technique.

Another problem with the design of this study is that duration of therapist contact was far longer in the combination and cognitive therapy groups than in the pharmacotherapy groups. It is thus not clear whether the effects observed for combination treatment and cognitive therapy were due to the specific components of the cognitive treatment (working through an interaction with drugs or on its own), or to the substantial directive therapist contact given to the 'cognitive' patients.

It is true that greater therapist-patient contact could help to explain the good response seen for combination treatment and cognitive therapy alone. However, in designing psychotherapy outcome studies which employ a drug control group, it may not always be essential to balance the groups in terms of frequency of contact and therapy time, for, as Rush et al. (1977) note, 'the studies that established the efficacy of pharmacotherapy for depressed outpatients used a once-a-week minimal contact design' (p. 34).

This criticism may be countered also by findings which show the superiority of drugs over various forms of psychotherapy (e.g. Covi et al., 1974; Friedman, 1975). If the total amount of time spent with therapists were the only factor relevant to outcome, then the psychotherapies investigated in these studies should have been more effective than drugs in reducing depressive symptoms.

Marks (1978) makes the point that psychological treatments for depression which involve a great deal of therapist time can only be justified if the results are 'outstandingly and lastingly' (p.533) better than conventional treatment with drugs since the latter are more cost-

effective. The data reported here do not indicate that cognitive therapy, with or without drugs, is necessarily 'outstandingly' better than chemotherapy, but they do support the contention that both are effective ways of treating depression, with combination treatment offering the greatest hope in relieving symptoms. The question of whether cognitive therapy provides a more lasting effect than drugs remains to be determined. It is hoped that the follow-up part of this study will shed light on this important dimension of psychotherapy research.

The importance of interpersonal and psychological factors in the treatment of depression should not be underestimated. The issue of therapist-patient contact simply underscores the need to determine to what extent this variable influences outcome, whether the treatments being investigated are physical, psychological, or a combination of the two.

Some factors related to the expectational sets of the therapists and patients, particularly in general practice, and features of the treatment setting were discussed earlier. On this point, the question could be raised as to whether the therapists' motivation to administer cognitive therapy and the amount of enthusiasm conveyed in the therapy sessions may have had a strong nonspecific treatment effect. This criticism cannot be dealt with by trying to minimise the role of therapist motivation. Obviously, both psychologists were highly motivated to become 'cognitive therapists' as evidenced by the amount of effort put into learning the techniques, not to mention the fact that they were prepared to take on a caseload of depressed outpatients, many of whom were suicidal (see appendix XI) and treat a considerable number with psychotherapy only, which proved to be a demanding experience to say the least. On the other hand, as

in studies which reported the superiority of cognitive therapy over other kinds of psychological treatment (e.g. Shaw, 1977), the therapists, being based on a pharmacological unit, had no a priori commitment to cognitive therapy. It is important to remember, too, that there is no reason to believe that a consultant psychiatrist (or his team of highly motivated and pharmacologically-oriented registrars) would be any less enthusiastic about the potentially dramatic effect of drug therapy combined with supportive psychotherapy.

Although enthusiasm and dedication to cognitive therapy may have contributed to the effects observed for cognitive therapy alone and combination treatment, this variable further supports the argument for more research into the relationship between therapist qualities, interactional processes in psychotherapy, and outcome (Parloff et al., 1978).

Turning to general problems related to measurement, this treatment study fulfilled an important requirement of any therapeutic trial by including independent assessments of patients in the different treatment groups. However, as in many studies of this kind, the results rest heavily on self-ratings. These measures are problematic in that they might be especially prone to change with cognitive behavioural treatments which might be effective in training the patient in new cognitive styles, that are simply reproduced in self-ratings but which are not generated by less verbally-directive control conditions, such as pharmacotherapy and non-specific, supportive psychotherapy. It is thus not clear, for example, whether the quicker response rates seen for combination treatment and cognitive therapy (BDI, HS) were due to the actions of each treatment, or to the fact that patients in these groups by virtue of their exposure to the 'cognitive therapy style', had been verbally conditioned to respond

in a way that favoured psychotherapy. It is difficult to refute this argument as cognitive therapy is geared in part to focusing on and reducing verbalised affective pain (and its associated cognitions) and hopelessness in particular.

Differential response set and the effect it has on self-ratings is an important factor which must be considered in the interpretation of the results of this study. To control for this variable, future studies should employ, in addition to self-rating scales, behavioural measures of depression such as that used by Dunn (1979, see p.) which would be perhaps less sensitive to contaminating effects of response set. An alternative way to analyse the behavioural measures used in this study, i.e. intra-individual correlations with Z score transformations for group comparisons, might also yield more convincing results and provide data that might offset the problem of response set in self report scales.

Another difficulty with the present investigation and this is true for many treatment studies (Kazdin and Wilson, 1978), is that the outcome criteria used may not be totally adequate for assessing the effects of the different treatments. Group differences, in terms of magnitude of mean change in reducing depressive symptoms, provide an extremely limited criterion for comparing treatments or for evaluating a given treatment. While an additional criterion was used in this study (i.e. the proportion of patients who improved markedly), several other related criteria need to be considered in future short-term studies. These should include, for example, the clinical significance or overall importance of the improvement effected in the individual patient (i.e. does improvement enhance the patient's everyday functioning in such areas as employment and social and married life?). Another criterion suggested by Kazdin and Wilson (1978)

would be breadth of change, or range of therapy effects (e.g. does 'improvement' mean that the patient is better able to cope with problems outside of the direct therapeutic focus on the defined problem?). One way to assess this might be to have the patient monitor life events on a regular basis over the course of treatment, identify these as desirable or undesirable, and then rate how effectively he thinks he has been able to deal with the undesirable events. This might also help the clinician to determine to what extent changes in symptoms, for example, were due to the treatments given or factors outwith the treatment setting.

The concurrent validity of the measures, while far from what was hoped for was satisfactory except for the behavioural measures. Of special interest were the findings on the semantic differential. The highly significant correlations between the concepts relevant to Beck's theory (views of self and world = .60; self and future = .58; and world and future = .51, all at $p = < .001$) provide further indirect support for a major component of the cognitive model of depression.

Criticisms which have been leveled against the semantic differential, e.g. lack of internal consistency of scales alleged to be measuring a single factor, and a high degree of concept-scale interaction (Warr and Knappen, 1965; Presley, 1969), do not apply to the use of the semantic differential made in this study as no attempt was made at this point to work out factorial dimensions of the scale. The bipolar adjectives used were, on the whole, those clinically used to elicit the three factors of semantic differential analysis, i.e. evaluation, potency and activity. These bipolar scales were scored only for their positive and negative prime facie characteristics. Future studies are planned which set out to examine the factorial dimensions of the scales.

Finally, an obvious flaw in the present research is the small size of the sample, particularly in the general practice setting where there were less than 10 patients in each group. It is possible that the outcome of this study would have been quite different had larger numbers of subjects been involved. A large sample might further detect between-group differences in post-treatment scores.

In spite of the shortcomings of the current investigation, the results have numerous practical applications and highlight areas for future research. Given further cross validation of the present findings with larger samples, combined cognitive therapy plus drugs and cognitive therapy alone may become recognised as effective ways of treating moderately depressed hospital and general practice patients.

I. SOME INTERPRETATIONS AS TO WHY COGNITIVE THERAPY WORKS

One major finding of this study is that each treatment was effective in alleviating depression, yet why this was so is not clear.

Most of the current theoretically derived psychological therapies assume that depressed patients show specific competence deficits and treatment addresses the problem thought to be most critical. Beck (1976) sees depressed patients as manifesting cognitive deficits (e.g. negative cognitive triad): treatment is aimed at changing negative thoughts (Rush et al., 1977). Fuchs and Rehm (1977) see self-control as a critical problem and aim therapy at restoring self-control. Grof (1977) has demonstrated that increasing mood-related pleasant activities is more effective than self-monitoring mood related activities or increasing non-mood related activities.

These different approaches to enhancing a specific competency have been effective in reducing depression. The assumption behind all of these studies is that therapeutic results were obtained because the treatment

was effective in selectively influencing the specific target behaviour. Zeiss et al. (1979) point out that with few exceptions these investigations did not show that the target behaviours (e.g. negative thoughts, or low rates of self-reinforcement) were selectively affected. Rather, they showed only that level of depression was reduced as predicted. The present study demonstrated that cognitive variables were affected by the cognitive treatments but this occurred for pharmacotherapy as well. How can these data be explained?

With regard to cognitive therapy, one explanation could be that treatment did, in fact, change depression-inducing cognitions. This contention receives support from a recent study by Rush et al. (1979). They found that, while both cognitive therapy and drug therapy significantly reduced hopelessness and improved self-concept, cognitive therapy resulted in more marked improvements in self-concept. Moreover, cognitive therapy was associated with an earlier and greater impact on hopelessness than pharmacotherapy, a finding borne out by the present study. These data suggest that cognitive therapy may have had a specific effect on the target for change, i.e. negative thoughts.

An equally plausible explanation has been put forward by Bandura (1977). He argues that whether a psychological therapy aims for specific or more global changes (e.g. Kelly, 1955; Rogers, 1970), it must change the patient's sense of personal efficacy, i.e. the expectation that he can perform the behaviours necessary to achieve desired outcomes. This, according to Bandura, is often best accomplished through performance-based procedures with a relatively specific focus. By learning to cope effectively with a problem, the patient becomes more confident in his ability to handle life's problems.

Bandura's self-efficacy model has received indirect support in an interesting study concerned with nonspecific improvement effects in the treatment of depression. Zeiss et al. (1979) treated depressed outpatients with therapies which focused on either interpersonal skills, negative cognitions or pleasant events. The results indicated that all treatments were successful in alleviating depression but none had treatment-specific effects on a single class of behaviour. The authors concluded, 'the treatments affected depression because all treatments provided training in self-help skills, thus increasing the patient's expectations of mastery and encouraging the perception of greater positive reinforcement as a function of the patient's greater skillfulness. This effect was independent of the specific training offered in any treatment module' (p. 438).

Other explanations concerning the efficacy of psychotherapy are grounded in research related to the study of nonspecific treatment variables thought to characterise all therapies - patient-therapist roles, expectations of improvement, therapy 'rituals' (Bergin and Suinn, 1975; Gomez-Schwartz, et al., 1978).

Pharmacotherapy may influence cognitive factors in a less direct way than specific psychotherapies. It may be that drugs, through intervention at the biochemical level (Akiskal and McKinney, 1975), restores the homeostatic balance thought to be disrupted by depressive illness, with cognitive changes occurring secondarily as the organism achieves a greater degree of physiological equilibrium.

Combined cognitive therapy and drugs would operate presumably at both the biological and psychological levels simultaneously. This multi-level action might account for the somewhat superior effect observed for combinatio

treatment in the hospital group. Against this theoretical framework, the equivalent effect seen for combination treatment and cognitive therapy in general practice is hard to explain. Factors related to compliance and the expectations of both the therapists and patients in the general practice setting and sample size could account for the lack of a superior effect for combination treatment.

It is worth emphasising, in summary, that this research, being an outcome study, has no pretensions of delineating therapeutic processes. The ideas discussed in this section are largely speculative, although they are based on some observations.

CONCLUSIONS AND IMPLICATIONS

A. The present study has concentrated upon examining the scope and limitations of cognitive therapy alone and in combination with pharmacotherapy in the treatment of depressed outpatients. The attempt to utilise cognitive therapy to treat moderately depressed hospital and general practice patients has largely substantiated the expectations arising from previous research in which this form of psychotherapy has been found to be effective. Thus, cognitive therapy appears to be a strong contender as one of the most effective interventions for the treatment of clinical depression. For hospital outpatients it is at least as effective as pharmacotherapy and, for responders as a group, it appears to provide more rapid improvement than pharmacotherapy in reducing both affective and cognitive symptoms of depression. Cognitive therapy seems to be a particularly powerful technique for decreasing hopelessness, a cognitive feature of depression which has been shown to predict suicide.

B. The findings from the sample of patients treated in Leith indicate that cognitive therapy may be more effective than pharmacotherapy in the treatment of the sort of depression seen in an urban family practice. Moreover, it appears as though cognitive therapy has the potential to attain parity with traditional methods of behaviour therapy in terms of its applicability to a broad range of patients. This study has demonstrated that patients from lower social class, having only a basic level of education, can learn the cognitive therapy style. This suggests, therefore, that some of the difficulties presented by socially disadvantaged people can be handled in this way.

C. The results discussed in this thesis extend the limited findings of previous research concerned with the use of psychological treatments in conjunction with pharmacotherapy. From a clinical viewpoint, combined cognitive therapy and pharmacotherapy appearsto be more effective than either treatment alone in reducing overall symptoms in hospital out-patients suffering from chronic, nonpsychotic depression. The effect for combination treatment appears to be additive in this population in the same way as Weissman et al. (1979) found interpersonal psychotherapy and pharmacotherapy to be superior to either on its own. For those who respond to therapy, combination treatment gives the added advantage of a much quicker and sharper effect, in particular on hopelessness.

D. The finding that cognitive therapy with or without drugs is associated with an earlier and greater impact on hopelessness than pharmacotherapy in responders may have substantial clinical meaning for the initial focus (i.e. hopelessness) of psychotherapy with depressed patients.

Moreover, it may help to explain why patients who show an initial response to drug therapy are at some increased risk of committing suicide (e.g. Silverstone, 1974). That is, hopelessness and suicide intent may remain high while motivation, through the onset of drug effects, is increased. In these circumstances patients may be more inclined to act on their intentions to harm themselves during this stage of pharmacotherapy. Thus, cognitive therapy may be particularly better than the supportive psychotherapy given with medication because of its specific early impact on hopelessness.

E. For the less chronic general practice group, the equivalent effect observed for combination treatment and cognitive therapy alone suggests that drugs do nothing to enhance the effect of cognitive therapy administered on an individual basis to patients in the doctor's surgery. It would appear that if patients are to be treated for clinical depression in general practice then cognitive therapy stands out as a viable alternative to chemotherapy. The fact that cognitive therapy is less time consuming than traditional psychotherapies and is feasible for a wide range of patients make it very suitable for use in the general practice setting.

F. If the criterion of ease of dissemination of therapeutic technique is invoked, then cognitive therapy fares particularly well compared to traditional forms of psychotherapy which require extensive training, supervision and experience. Using the teaching materials supplied by Beck and his associates, the therapists involved in this study were able to learn and practice cognitive therapy with a fair degree of success. Thus, cognitive therapy appears to be a very teachable and easily disseminated

type of psychological treatment.

G. The results discussed in this thesis, as well as the author's day-to-day experience in the clinic, argue strongly for a close collaboration between the descriptive diagnostician, the biologically oriented psychiatrist (or G.P.) and the behaviourally oriented psychotherapist. In short, it appears that pharmacotherapy and psychotherapy cannot be regarded as 'competitors' in the management of moderately depressed outpatients. Rather, it would be more constructive to think of these treatments as 'allies', since this research provides tentative evidence that different kinds of patients will respond to either drugs or cognitive therapy or a combination of the two. The findings also provide further indirect support for a multifactorial hypothesis in depression - hence for the management of this disorder.

H. Whether <u>short-term</u> cognitive therapy with or without drugs offers any special advantages over drugs alone in the prophylaxis of depression is an important question. This hypothesis, however, can be tested only through lengthy follow-up investigations which compare the relative relapse rates of patients who received combination treatment, cognitive therapy, or pharmacotherapy. The follow-up stage of this project may help to address this crucial issue.

I. Some implications of the obtained findings for future research have already been discussed. This study does not provide enough data to say definitely that any one treatment modality in the hospital group is statistically superior to any other, a problem which may be due to the large variances in each treatment group. Similar studies that include

larger and more homogenous samples might contribute more in the way of statistical evidence that could support the significant clinical findings obtained for the hospital outpatients in this study.

The multiple regression analysis used in this study allows for the testing of specific hypotheses as regards the kind of people who are likely to respond to combination treatment, cognitive therapy and drugs (p. 314, and appendix XIX). Future research could be directed towards examining whether the predictor variables obtained in this study are, in fact, valid predictors of outcome.

No definite statement can be made about the efficacy of combination treatment or cognitive therapy alone unless there is knowledge of outcomes of similar patients receiving alternative psychological treatments (e.g. self-control therapy, interpersonal psychotherapy). This would help to establish which of the psychological therapies were most beneficial and, given the use of multiple outcome criteria, could shed light on the specific effects of the different treatments.

Future clinical trials of cognitive therapy should also include 'inert treatment' groups (e.g. attention-by-assessment) to control for the nonspecific effects of attention, as well as other ethically feasible conditions (e.g. low contact, the nonscheduled treatment method proposed by Weissman et al., 1979) which could be used to determine whether the natural history of the depressive disorder can be influenced by even minimal intervention, let alone the treatments under investigation.

Since cognitive therapy is an amalgam of behavioural and cognitive techniques (e.g. graded tasks, assertion training, cognitive restructuring) much work needs to be done in teasing out the critical components of the cognitive therapy package.

Another concern for future research is to establish whether cognitive therapy, particularly when used in conjunction with drugs, has anything to offer severely ill depressed patients, such as psychotic, hospitalised or bipolar patients.

There is a need to decide whether cognitive therapy has any beneficial effect on general social adjustment and whether personality factors, especially enduring attitudes, may be influenced by this form of treatment.

Finally, more research is needed to try and incorporate the various approaches subsumed under the label 'cognitive therapy' into one consistent theoretical and treatment model.

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APPENDIX I

Hourly diary of waking activities

WEEKLY ACTIVITY SCHEDULE

NOTE Grade activities M for mastery and P for pleasure

	M	T	W	Th	F	S	S
9-10							
10-11							
11-12							
12-1							
1-2							
2-3							
3-4							
4-5							
5-6							
6-7							
7-8							
8-12							

APPENDIX II

Triple column technique

DATE	Situation Describe: 1. Actual event leading to unpleasant emotion, or 2. Stream of thoughts, daydream, or recollection leading to unpleasant emotion	Emotion(s) 1. Specify sad/ anxious/angry, etc. 2. Rate degree of emotion, 1-100	Automatic thought(s) 1. Write automatic thoughts that preceded emotion(s) 0-100%	Rational response 1. Write rational response to automatic thought(s) 2. Rate belief in rational response 0-100%	Outcome 1. Re-rate belief in automatic thought(s), 0-100% 2. Specify and rate subsequent emotions, 0-100

EXPLANATION: When you experience an unpleasant emotion, note the situation that seemed to stimulate the emotion

APPENDIX III

Cognitive therapist competency checklist

Steve Bishop

General Comments:

Interpersonal factors and basic clinical skills appeared excellent. Interest, warmth, sincerity, and especially enthusiasm were strong points. Session was clearly within cognitive ball park (specific qualifications will follow). There was some deficit in structural elements. An overt, stated agenda was absent (although it was clear one was held in mind). The session seemed to generally go with the flow of conversation, rather than follow a specific structure. Balance between collaboration and didactically supplying cognitions or responses was a bit too weighted toward latter. Overall, session was quite good.

Specific Comments: (loosely follows content and sequence of tape)

- Checking on events of past week good (although better in context of a complete agenda. The implicit agenda appeared to be:

1) events of past week
2) jobs
3) guilt: a) abortion
   b) separation
4) social relations: a) men
   b) women

It would have helped had this been made explicit at start of session.

- Good positive reinforcement regarding patient's going through union channels with her complaint. Empathetic comments good--acknowledging her feelings and the difficulties of her position. Self-disclosure--good.

- Portion concerning drunk women at bar at patient's second job--"How'd you handle it"--good, but primarily a behavioral question. Should have been coupled with cognitive question--". . . and what thoughts did that bring to mind?"

- When patient said, "I was lazy," important not to let this slip by. Define as symptom rather than personality trait.

"Bad feelings of husband having chosen someone else"--good in getting out associated cognitions and assumptions. Might have defined in more concrete way--listing reasons related to her for husband going elsewhere and reasons unrelated to her--thereby dealing with self-attribution.

Having patient "convince" you her good points as a wife--a good technique. Re-attribution effectively accomplished by listing his bad qualities, or at least referring to them.

"I can't stand swearing . . . gets me edgy." What are the specific cognitions related. What intervenes to make this a noxious stimulus. Couple behavioral approach, e.g., distraction (getting behind bar and turning attention to something else) with cognitive approach.

Eliciting specific cognitions about abortion well done--especially in defining "cruel" and pointing out difference between cruel person and doing a cruel act. Using idea of cruel-kind continuum good. Might have introduced concept of percentage to counter all-or-nothing aspects.

"If were coming to you with this problem . . ."--good decentering technique.

Be aware of dangers of confronting a conviction (religious, moral, political, etc.) directly. Taking a pragmatic approach (as eventually done) via looking at consequence is more effective. Good in accepting her difficulties in this area (i.e., abortion) and allow her to express some bad feelings.

Feedback on assigned homework--very good as was progress review of session and course of therapy and review and writing down current homework.

Rev. 2/1-11-79 JY

Competency Checklist for Cognitive Therapists

erapist B. Stagg Patient _____ Date of Session _____

ter Freeman / / One-way mirror / / Videotape / ✓ Audiotape

te of Rating _____

. Collaboration and Mutual Understanding

✓ a. Therapist worked with patient even when using primarily an educative role.

✓ b. Therapist asked for feedback.

✓ c. Patient gave feedback.

_ d. Therapist asked for suggestions (or offered choices).

_ e. Patient offered suggestions (or made choices).

✓ f. Therapist responded to patient's feedback and/or suggestions; did not ignore or negate them.

_ g. Therapist checked periodically for understanding of key points made by patient.

✓ h. Therapist spoke freely and seemed comfortable with the patient.

✓ i. The patient spoke freely and seemed comfortable with the therapist.

. Questioning

✓ a. Therapist asked questions frequently.

✓ b. Some questions were "open-ended."

✓ c. Some questions were inductive; they helped the patient arrive at a particular conclusion or test a particular hypothesis.

✓ d. Some questions were asked to gather data.

✓ e. Therapist did not "cross-examine" the patient; "trap" the patient, or make the patient try to guess what the therapist has in mind.

. Established Agenda (not applicable for first session)

_ a. Therapist established agenda for session.

_ b. Agenda items were specific and problem-oriented, rather than vague or general topic areas.

✓ c. At some point, therapist discussed events during the week since the last session.

_ d. Priorities for agenda items were established.

_ e. Agenda was appropriate for time allotment (neither too ambitious nor too limited)

. Elicits Feedback

Therapist elicits patient's feelings and reactions to:

✓ a. Present interview.

✓ b. Previous interview.

5. Structures Therapy Time Efficiently

0 a. Therapist covered most items on agenda and rescheduled unfinished business.

✓ b. Therapist was flexible enough to include important issues that arose during session but were not on the agenda.

– c. Therapist stopped or limited time spent on peripheral or tangential topics.

0 d. Therapist stopped or limited unproductive discussion on relevant topics.

6. Focuses on Appropriate Problem

✓ a. Therapist identified specific problem(s) to focus on.

✓ b. Identified problems were central to patient's distress rather than peripher

✓ c. Identified problems were appropriate for treatment at this time.

✓ d. Identified problems were the key ones to focus on; the major problem was not overlooked.

✓ e. Therapist concentrated on one or two problems instead of skipping around.

7. Elicits Automatic Thoughts

✓ a. Specific automatic thoughts were identified.

✓ b. Therapist helped patient identify thoughts rather than repeatedly point out automatic thoughts to patient in a didactic fashion.

✓ c. Therapist used appropriate techniques to elicit automatic thoughts (circle techniques used):

(inductive questioning) (mood shifts during session)

imagery (dysfunctional thought record)

role-playing

✓ d. Therapist helped patient recognize connection between affect and specific cognitions.

8. Tests Automatic Thoughts

✓ a. Therapist tested or questioned automatic thoughts in some manner.

✓ b. Therapist did not rely solely on didactic approaches (i.e., pointing out irrationality).

✓ c. Therapist helped patient set up specific, testable hypotheses.

✓ d. Therapist helped patient collect valid evidence systematically concerning hypotheses.

✓ e. Therapist helped patient evaluate evidence and draw conclusions from evidenc

9. Identifies and Tests Underlying Assumptions

- ✓ a. Specific underlying ("silent") assumptions were identified.
- ✓ b. Therapist helped patient discover relevant assumptions from a joint analysis of automatic thoughts.
- ✓ c. Therapist did not rely solely on didactic counterarguments to evaluate assumptions.
- ✓ d. Therapist helped patient analyze validity of assumptions (e.g., by inductive questioning or by listing advantages and disadvantages).

10. Selects Appropriate Techniques

- ✓ a. Techniques used were generally appropriate for identified problems.
- ✓ b. Techniques used were the most appropriate for identified problems (e.g., preferable techniques were not overlooked).
- ✓ c. Therapist executed techniques successfully.
- d. Specific materials, instruments, and devices:

___ activity schedule: summary
___ activity schedule: planning
___ mastery and pleasure ratings
✓ list of main points for patient
___ reading assignment
✓ daily record of dysfunctional thoughts
___ wrist counter
___ videotape of session for patient
___ audiotape of session for patient
___ "walkie-talkie"
___ questionnaires to elicit new material (e.g., DAS, BDI, YLI)

- e. Techniques used:

___ cognitive rehearsal
___ role-playing
___ distraction procedures
___ self-reliance training
___ sensory awareness exerciese
___ relaxation exercises
✓ reattribution
___ alternative therapy
___ autobiographies

11. Provides Progress Summaries

- ✓ a. Therapist periodically recapitulated or reformulated problems being worked on session.
- ✓ b. Therapist explained rationale for techniques or approaches used during session.
- ✓ c. Therapist summarized progress made on identified problems during the session (problem closure).
- ___ d. Therapist assessed (for patient's benefit) progress thus far in therapy and future plan.
- ✓ e. Therapist and patient achieved consensus regarding therapy progress.

2. Assigns Homework

0 a. Therapist carefully reviewed previous week's homework.

b. Therapist summarized conclusions derived, or progress made, from previous homework.

✓ c. Therapist assigned new homework.

✓ d. Homework assignment was appropriate for identified problems.

✓ e. Therapist explained rationale for homework assignment.

✓ f. Homework was specific and details were clearly explained.

✓ g. Therapist asked patient if he/she anticipated problems in carrying out homework

h. Therapist integrated patient's suggestions, if any, into the assignment.

3. Genuineness

✓ a. Therapist seemed to be saying what he sincerely felt. Seemed honest and "real."

✓ b. Therapist seemed open rather than defensive.

✓ c. Therapist did not seem to be holding back impressions or information, or evading patient's questions.

✓ d. Therapist did not seem patronizing or condescending. Treated the patient as an "equal."

✓ e. Therapist did not seem to be playing the role of a therapist. Did not sound contrived or rehearsed.

4. Warmth

✓ a. Therapist's tone of voice and non-verbal behavior conveyed warmth and interest.

✓ b. The content of what the therapist said communicated concern and caring.

✓ c. The therapist did not criticize, disapprove, or ridicule the patient's behavior.

✓ d. The therapist did not seem cold, distant, or indifferent.

✓ e. The therapist did not seem effusive, possessive, or over-involved.

✓ f. The therapist responded to or displayed humor when appropriate.

5. Accurate Empathy

✓ a. The therapist accurately summarized what the patient explicitly said.

✓ b. The therapist accurately summarized the patient's most obvious emotions (e.g., sadness, anger).

✓ c. The therapist accurately summarized more subtle nuances of feeling or implicit belief.

✓ d. The therapist communicated through his/her voice that he/she sensed the patient's feelings and was responding to them.

✓ e. The therapist did not inaccurately project his/her own problems, conventional "wisdom" or attitudes derived from a particular theoretical system onto the patient.

6. Professional Manner

✓ a. Therapist's tone of voice and non-verbal behavior (e.g., posture) conveyed confidence.

✓ b. Therapist made clear statements without frequent hesitations or rephrasings.

✓ c. Therapist took sufficient control of the session; did not seem too passive by allowing patient to dominate session.

✓ d. Therapist's appearance was "professional."

✓ e. Therapist seemed relaxed and did not seem to be anxious or "trying too hard."

7. Rapport

Good!

APPENDIX IV

Present State Examination

Present State Examination

J. K. WING, J. E. COOPER and
N. SARTORIUS

CAMBRIDGE UNIVERSITY PRESS

PRESENT STATE EXAMINATION

(Ninth edition of interview schedule, May 1973)

Project no. 1 2

Subject no. 3 4 5

Card no. 6 7 8 9 10

Interviewed at:

Interviewed by:

Date and time of interview:

Type of service or setting:

Date of admission, etc.:

Name of agency:

Interview rated by:

Date of rating:

Live_____ V/T_____ A/T_____ Film_____

Episode no.

MRC Social Psychiatry Unit
Institute of Psychiatry
London
SE5 8AF

INSTRUCTIONS

The instruction manual contains a detailed description of the origins, development and underlying principles of the PSE and a glossary of definitions of symptoms. The examiner must be thoroughly familiar with the manual and glossary and should have had some prior training in the use of the PSE.

Four kinds of question are written into the schedule:

(*a*) *Obligatory (starred) questions*

These must be asked if the interview is conducted at all. Only 54 questions are involved. Thus subjects with no symptoms, who ask clarifying questions of their own and who answer clearly and decisively, can be screened very quickly indeed. Whenever there is any doubt, however, and certainly whenever a symptom needs clarification, the second kind of question should be asked.

(*b*) *Bracketed questions above cut-off points*

These help to define the nature and extent of a symptom and should always be asked if there is any doubt about replies to obligatory questions.

(*c*) *Unbracketed questions below cut-off points*

Once the examiner has proceeded below a cut-off point, he must ask *all* the unbracketed questions in that part of the section.

(*d*) *Bracketed questions below cut-off points*

These serve the same function as similar questions above cut-off points, i.e. they help to define the nature and extent of a symptom. They are used only if there is some other evidence that the symptom is present.

In addition, the examiner himself will usually wish to ask other questions which are not written into the schedule, either general probes or more specific questions, depending on the nature of the patient's replies.

Each symptom is defined to some extent within the schedule itself but the examiner must be completely familiar with the fuller definitions in the glossary. A full discussion of scoring is also included in the glossary, particularly as to how to differentiate (0) from (1), and (1) from (2).

(0) = Examiner satisfied that symptom not present to clinically significant degree during past month.

(8) = Examiner not sure whether symptom present during past month, even though the appropriate questions have been asked, and answered without incoherence or evasion. The symptom cannot be excluded.

(9) = No rating can be made because question not asked or subject does not answer or answer is incomprehensible.

It should be emphasised that using the PSE schedule will not in itself guarantee useful results. The quality of the output of any system depends on the quality of the input.

1. INTRODUCTION

The interviewer should introduce himself briefly, describe the purpose of the interview and explain about any recording equipment. The purpose of the introductory section is to obtain an overall picture of the symptomatology, in the subject's own words.

** To begin with, I should like to get an idea of the sort of problems that have been troubling you during the past month. What have been the main difficulties?

Record the main symptoms spontaneously mentioned.

Means of exploration, if subject gives inadequate information:

If subject's statement too brief	Can you tell me more about that?
If subject has no more to add	What else has been troubling you?
If statements are difficult to understand	Can you explain what you mean by . . . ?
If subject is vague	Could you give an example of . . . ?
If no other response forthcoming	Why did you come to the (hospital)?

RATE PATIENT'S ACCOUNT OF SYMPTOMS.

0 = Subject responds adequately.

1 = Account somewhat inadequate but interview can proceed.

2 = Account seriously inadequate but interview proceeds in an attempt to rate some subjective responses, as well as behaviour, affect and speech. _____ (see 140)

3 = Impossible to continue with interview. Only behaviour, affect and speech sections rated.

REASONS FOR INADEQUACY (TICK AS MANY AS APPROPRIATE).

Denial or guardedness	_____	Inattention	_____
Incoherence	_____	Refusal	_____
Irrelevance	_____	Patient mute, stuporous, etc.	_____
Replies too brief	_____	Other, specify	_____
Poverty of content of speech	_____		

IF (1) OR (2) CARRY ON WITH SECTION 2, UNLESS SUBJECT MENTIONS OR HINTS AT DELUSIONS OR HALLUCINATIONS → SECTION 18.

Cut off

Current treatment, if subject not seen in hospital or clinic
Rate the following if sufficient information has already emerged.
If not, use the suggested question:
May I ask if you are seeing any doctor for your nerves?
Or specify if psychosomatic complaints.

What kind of doctor is he?
Your own GP? A private doctor? Psychiatrist?

0 = No doctor
1 = GP
2 = Private doctor other than GP
3 = Psychiatrist
4 = Hospital out-patient (other than psychiatric)
5 = Other paramedical specialist, or osteopath
6 = Other specify

Are you attending for treatment any person who is not medically qualified, e.g. lay therapist, herbalist, acupuncture, faith healer, Christian Science, church which forbids medical advice?
What were you complaining of at the time?

Specify type of treatment

Complaint

2. HEALTH, WORRYING, TENSION

** Is your physical health good?
(Does your body function normally?)

** Do you feel you are physically ill in any way?
(What is that like? How serious is it?)

RATE SUBJECT'S OWN SUBJECTIVE EVALUATION OF PRESENT PHYSICAL HEALTH (*irrespective of whether physical disease is present*). ☐ (1)

0 = Feels physically very fit.
1 = Feels particular physical complaint but does not say positively feels fit.
2 = Feels unwell but not seriously incapacitated.
3 = Feels seriously incapacitated by physical illness.

** What does your doctor say is wrong?
(Have you had a physical illness recently; colds, influenza, etc.?)

RATE PRESENCE OF PHYSICAL ILLNESS OR HANDICAP, *taking results of recent investigations and physical state examinations into account.* ☐ (2)

0 = No physical illness or handicap present.
1 = Mild but significant physical illness or handicap (e.g. influenza or limp).
2 = More serious physical illness or handicap present but not incapacitating or threatening to life (e.g. deafness or duodenal ulcer).
3 = Physical illness or handicap present which is incapacitating or threatening to life (e.g. blindness or carcinoma).

Specify illness, disabilities and duration:

RATE PSYCHOSOMATIC SYMPTOMS. *Special projects only* ☐ (3)

** Have you worried a lot during the past month?
(What do you worry about?)
PROBE: (Money, housing, children, health, work, marriage, relatives, friends, neighbours, other).
(How much do you worry? Are you a worrier?)
If any indication of worry, use further probes:

** What is it like when you worry?
(What sort of state of mind do you get into?)
(Do unpleasant thoughts constantly go round and round in your mind?)
(Can you stop them by turning your attention to something else?)
RATE WORRYING: *A round of painful thought which cannot be stopped and is out of proportion to the subject worried about.* ☐ (4)

1 = Symptom definitely present during past month, but of moderate clinical intensity or intense less than 50% of the time.
2 = Symptom clinically intense more than 50% of the month.

** Have you had headaches, or other aches or pains, during the past month?
(What kind?)
RATE ONLY TENSION PAINS, e.g. *'band round head', 'pressure', 'tightness in scalp', 'ache in back of neck', etc., not migraine.* ☐ (5)

1 = Symptom definitely present during past month, but of moderate clinical intensity, or intense less than 50% of the time.
2 = Symptom clinically intense more than 50% of past month.

** Have you been getting exhausted and worn out during the day or evening, even when you haven't been working very hard?
RATE TIREDNESS OR EXHAUSTION: *Do not include tiredness due to 'flu, etc. = 9.* ☐ (6)

1 = Only moderate form of symptom (tiredness) present; or intense form (exhaustion) less than 50% of the time.
2 = Intense form of symptom (exhaustion) present more than 50% of the past month.

** Have you had difficulty in relaxing during the past month?
(Do your muscles feel tensed up?)
RATE MUSCULAR TENSION: *Do not include a subjective feeling of nervous tension, which is rated later.* ☐ (7)
1 = Symptom definitely present during past month, but of moderate clinical intensity, or intense less than 50% of the time.
2 = Symptom clinically intense more than 50% of past month.

** Have you been so fidgety and restless that you couldn't sit still?
RATE RESTLESSNESS. ☐ (8)
(Do you have to keep pacing up and down?)
1 = Only moderate form of symptom (fidgety, restless) present; or intense form (pacing, can't sit down) less than 50% of the time.
2 = Intense form of symptom (pacing, etc.) present more than 50% of past month.

** Do you tend to worry over your physical health?
RATE HYPOCHONDRIASIS: *Overconcern with possibility of death, disease or malfunction. Re-rate at end of interview if subject constantly reverts to hypochondriacal preoccupation. Consider ratings of symptoms (1) and (3).* ☐ (9)
1 = Symptom present during past month, but not (2).
2 = Subject constantly reverts to hypochondriacal preoccupations during interview.

** Do you often feel on edge or keyed up or mentally tense or strained?
(Do you generally suffer with your nerves?)
(Do you suffer from nervous exhaustion?)
RATE SUBJECTIVE FEELING OF 'NERVOUS TENSION': *There is no need for autonomic accompaniments for this symptom to be rated present.* ☐ (10)
1 = Symptom definitely present during past month, but of moderate intensity, or intense less than 50% of the time.
2 = Intense form of symptom present more than 50% of the past month.

** Do you find that a lot of noise upsets you?
(Do noises sometimes seem to penetrate, or go through your head?)
RATE HYPERSENSITIVITY TO NOISE. ☐
1 = Moderate degree during month.
2 = Severe degree during month.

3. AUTONOMIC ANXIETY

In this section, rate only subjective anxiety with autonomic accompaniments, either free-floating or situational. Do not include worrying or nervous tension. Do not include anxiety due to, e.g., persecutory delusions, except in the special item (no. 13).

(CHECK LIST of autonomic accompaniments:

Blushing	Dry mouth
Butterflies	Giddiness
Choking	Palpitations
Difficulty getting breath	Sweating
Dizziness	Trembling)

** Have there been times lately when you have been very anxious or frightened?
(What was this like?)
(Did your heart beat fast?) *Ask for other autonomic symptoms.*
(How often in the past month?)

RATE FREE-FLOATING AUTONOMIC ANXIETY: *Exclude if due to delusions. Exclude if purely situational.* ☐ (11)

1 = Symptom definitely present, with autonomic accompaniment, during past month, but of moderate clinical intensity, or intense less than 50% of the time.
2 = Symptom clinically intense more than 50% of the time.

** Have you had the feeling that something terrible might happen?
(That some disaster might occur but you are not sure what? Like illness or death or ruination?)
(Have you been anxious about getting up in the morning because you are afraid to face the day?)
(What did it feel like?)

RATE ANXIOUS FOREBODING WITH AUTONOMIC ACCOMPANIMENTS. ☐ (12)

1 = Symptom definitely present, with autonomic accompaniment, during past month, but of moderate clinical intensity, or intense less than 50% of the time.
2 = Symptom clinically intense more than 50% of the time.

RATE AUTONOMIC ANXIETY DUE TO DELUSIONS, *etc. and if necessary defer to end of interview.* ☐ (13)

0 = No anxiety due to delusions or hallucinations.
1 = Subject complains of anxiety but no evidence of anxiety on examination.
2 = Clearly anxious or frightened because of delusions or hallucinations.

CUT OFF IF NO EVIDENCE OF ANXIETY OR IF ANXIETY DUE ONLY TO DELUSIONS → SECTION 4.

Cut off ☐

Have you had times when you felt shaky, or your heart pounded, or you felt sweaty, and you simply had to do something about it?
(What was it like?)
(What was happening at the time?)
(How often during the past month?)

RATE PANIC ATTACKS WITH AUTONOMIC SYMPTOMS:
A panic attack is intolerable anxiety leading to some action to end it, e.g. leaving a bus, phoning husband at work, going in to see a neighbour, etc. ☐ (14)

1 = One to four panic attacks during month
2 = Panic attacks five times or more.

Do you tend to get anxious in certain situations, such as travelling, or being alone, or being in a lift or tube train?
(What situations? How often during the past month?)
(CHECK LIST: *Can be presented on separate card and each item rated separately, if needed.*
Crowds (shop, street, theatre, cinema, church).
Going out alone; being at home alone.
Enclosed spaces (hairdresser, phone booth, tunnel).
Open spaces, bridges.
Travelling (buses, cars, trains).)

RATE SITUATIONAL AUTONOMIC ANXIETY. ☐ (15)

1 = Has not been in such situations during the past month but aware that anxiety would have been present if the situation had occurred.
2 = Situation has occurred during the past month and patient did feel anxious because of it.

What about meeting people, e.g. going into a crowded room, making conversation?
(CHECK LIST: *Present card if necessary:*
Speaking to an audience.
Eating, drinking or writing in front of other people.
Parties.)

RATE AUTONOMIC ANXIETY ON MEETING PEOPLE. ☐ (16)

1 = Has not been in such situations during the past month but aware that anxiety would have been present if the situation had occurred.
2 = Situation has occurred during the past month and patient did feel anxious because of it.

Do you have any special fears, like some people are scared of feathers or cats or spiders or birds?
(CHECK LIST: *Present card if necessary:*
Heights, thunderstorms, darkness.
Animals or insects of any kind.
Dentists, injections, blood, injury.)

RATE ONLY SPECIFIC PHOBIAS, NOT GENERAL SITUATIONAL ANXIETY. ☐ (17)

1 = Has not been in such situations during the past month but aware that anxiety would have been present if the situation had occurred.
2 = Situation has occurred during the past month and patient did feel anxious because of it.

Do you avoid any of these situations (specify as appropriate) because you know you will get anxious?
(How much does it affect your life?)
RATE AVOIDANCE OF ANXIETY-PROVOKING SITUATIONS. ☐ (18)

1 = Subject tends to avoid such situations whenever possible.
2 = Marked generalisation of avoidance has occurred during past month, e.g. subject has not dared to leave the house or has gone out only if accompanied.

Describe anxiety symptoms and list phobias.

4. THINKING, CONCENTRATION, ETC.

** Can you think clearly or is there any interference with your thoughts?

** Do your thoughts tend to be muddled or slow?
(Can you make up your mind about simple things quite easily?) (Make decisions about everyday matters?)
RATE SUBJECTIVELY INEFFICIENT THINKING *(if due to intrusion of alien thoughts, rate 9).* ☐ (19)

1 = Symptom definitely present during the past month, but of moderate clinical intensity, or intense less than 50% of the time.
2 = Symptom clinically intense more than 50% of the past month.

** What has your concentration been like recently?
(Can you read an article in the paper or watch a TV programme right through?)
(Do your thoughts drift off so that you don't take things in?)
RATE POOR CONCENTRATION. ☐ (20)

1 = Only moderate form of symptom present during the past month (e.g. can read a short article, can concentrate if tries hard); or intense less than 50% of the time.
2 = Symptom clinically intense (cannot attempt to read or concentrate) more than 50% of the past month.

** Do you tend to brood on things?
(So much that you even neglect your work?)
RATE NEGLECT DUE TO BROODING. ☐ (21)

1 = Symptom has caused moderate impairment to work or social relationships.
2 = Marked impairment.

** What about your interests, have they changed at all?
(Have you lost interest in work, or hobbies, or recreations?)
(Have you let your appearance go?)

RATE LOSS OF INTEREST *continuing during the past month.* ☐ (22)

1 = Symptom definitely present during the past month, but of moderate clinical severity or severe loss less than 50% of the time.
2 = Symptom clinically severe more than 50% of the past month.

** Have you become interested in new things at all?

IF EVIDENCE OF EXPANSIVE MOOD OR IDEAS → SECTION 9.

IF ODD IDEAS, EXPLORE FURTHER. PROCEED TO SECTION 15 IF APPROPRIATE.

** Have you suffered any lapses of memory recently? (PROBE ONLY)

IF EVIDENCE OF DISSOCIATION OR ORGANIC MEMORY LOSS → SECTION 16.

ANSWERS TO THESE QUESTIONS MAY SUGGEST THAT OTHER TYPES OF THOUGHT DISORDER ARE PRESENT, IF NOT, CUT OFF → SECTION 5.

Cut off ☐

IF ANY EVIDENCE OF THOUGHT DISORDER:

Are you in full control of your thoughts?
Can people read your mind?
Is anything like hypnotism or telepathy going on?

IF NECESSARY, PROCEED TO SECTION 13.

5. DEPRESSED MOOD

** Do you keep reasonably cheerful or have you been very depressed or low-spirited recently?
Have you cried at all?
(When did you last really enjoy doing anything?)

RATE DEPRESSED MOOD. N.B. *When rating clinical severity of depression remember that deeply depressed people may not necessarily cry. See definition in glossary.* ☐ (23)

1 = Only moderately depressed during past month, or deep depression for less than 50% of the time and tending to vary in intensity.
2 = Deeply depressed for more than 50% of the past month, and tending to be unvarying in intensity.

** How do you see the future?
(Has life seemed quite hopeless?)
(Can you see any future?)
(Have you given up or does there still seem some reason for trying?)
RATE HOPELESSNESS *on subject's own view at present.* ☐ (24)

1 = Hopelessness of moderate intensity but still has some degree of hope for the future (irrespective of time during month).
2 = Intense form of symptom (patient has given up hope altogether).

USE JUDGEMENT ABOUT WORDING.

** Have you felt that life wasn't worth living?
(Did you ever feel like ending it all?)
(What did you think you might do?)
(Did you actually try?)
RATE SUICIDAL PLANS OR ACTS. ☐ (25)

1 = Deliberately considered suicide (not just a fleeting thought) but made no attempt.
2 = Suicidal attempt but subject's life never likely to be in serious danger, except unintentionally.
3 = Suicidal attempt apparently designed to end in death (i.e. accidental discovery or inefficient means).

N.B. *Examiner should judge clinically whether there was intent to end life or not. If in doubt, assume not.*

Cut off ☐

IF EVIDENCE OF BOTH DEPRESSION AND ANXIETY RATE ANXIETY OR DEPRESSION PRIMARY.

If subject suffers from both anxiety and depression, and both have been rated as present, try to decide which is primary.
Which seems worse, the depression or the anxiety? (Use patient's own terms).

0. Anxiety is primary. Depression appears to be entirely explicable in terms of the limitations placed on the subject by the symptoms of anxiety, e.g. being unable to leave the house, travel, meet people, etc., or being afraid of heart disease because of palpitations. ☐ (26)
1. Anxiety and depression both present but seem independent of each other or it is not possible to decide whether one of them is primary.
2. Depression is primary. Anxiety is either a result of the depression (e.g. subject is frightened because of morbid or suicidal ideas) or it takes the form of fears of catastrophe, forebodings about illness or death, dread of having to face the day when first waking in the morning, preoccupation that something awful is going to happen. Panic attacks and situational anxiety, if present, are secondary to depression

Is the depression worse at any particular time of day?
RATE MORNING DEPRESSION *(particularly on waking)* (27)

0 = No depression.
1 = Not specially marked in mornings.
2 = Specially marked in mornings.

6. SELF AND OTHERS

** Have you wanted to stay away from other people?
(Why?)
(Have you been suspicious of their intentions? Of actual harm?)
RATE SOCIAL WITHDRAWAL. (28)

1 = Only passive form of symptom, i.e. subject does not seek company but does not refuse it if offered; or, if active withdrawal, less than 50% of the month.
2 = Actively avoids company (refuses it if offered). Actively withdraws in this way for more than 50% of the month.

** What is your opinion of yourself compared to other people?
(Do you feel better, or not as good, or about the same as most?)
(Do you feel inferior or even worthless?)
RATE SELF-DEPRECIATION. (29)

1 = Some inferiority, not amounting to feeling of worthlessness. If subject considers self to be worthless, this intense form of the symptom is present less than 50% of the time.
2 = Subject considers self to be completely worthless. Symptom present more than 50% of the month.

** How confident do you feel in yourself:
(For example, in talking to others, or in managing your relations with other people?)
RATE LACK OF SELF-CONFIDENCE WITH OTHER PEOPLE. *Consider only competence in social relationships, not competence at mechanical work, etc.* (30)

1 = Moderate lack of self-confidence, or intense lack less than 50% of the month.
2 = Intense lack of self-confidence more than 50% of the month.

** Are you self-conscious in public?
(Do you get the feeling that other people are taking notice of you in the street or a bus or a restaurant?)
(Do they ever seem to laugh at you or talk about you critically?)
(Do you consider people really are looking at you, or is it perhaps the way you feel about it?)
RATE SIMPLE IDEAS OF REFERENCE (NOT DELUSIONS). (31)

1 = Marked self-consciousness only (irrespective of time during month).
2 = Feels that people are criticising or laughing at self but can be reassured.

IF NO EVIDENCE OF GUILT, CUT OFF → SECTION 7.

(IF EVIDENCE OF MISINTERPRETATIONS, DELUSIONS OF REFERENCE OR PERSECUTION → SECTIONS 15B, 15C.)

Cut off

IF EVIDENCE OF GUILT:

Do you have the feeling that you are being blamed for something, or even accused? What about?

RATE GUILTY IDEAS OF REFERENCE. *Do not include justifiable blame or accusation. Exclude delusions of guilt.* (32)

1 = Subject feels blamed but not accused (irrespective of time during month).
2 = Subject feels accused of some sin or misdemeanour. Not delusional

IF DELUSIONS OF REFERENCE MAY BE PRESENT → SECTION 15B.

Do you tend to blame yourself at all?
(If people are critical, do you think you deserve it?)

RATE PATHOLOGICAL GUILT ONLY. (33)

1 = Subject feels over-guilty about some peccadillo (irrespective of time during month).
2 = Subject feels to blame for everything that has gone wrong even when not his fault, but not delusional.

IF DELUSIONS OF GUILT MAY BE PRESENT → SECTION 15G.

Do you blame anyone else for your troubles?

IF DELUSIONS OF PERSECUTION → SECTION 15C.

7. APPETITE, SLEEP, RETARDATION, LIBIDO

** What has your appetite been like recently?
(Have you lost any weight during the past three months?)

RATE LOSS OF WEIGHT DUE TO POOR APPETITE. (34)
Do not include changes due to physical illness.

1 = Less than 7 lb (15 kg).
2 = 7 lb (15 kg) or more.

** Have you had any trouble getting off to sleep during the past month?
(How long do you lie awake?)
(What happens if you take sleeping tablets?)
(How often does it happen?)

RATE DELAYED SLEEP. ☐ (35)
1 = One hour or more delay (irrespective of sleeping tablets).
2 = Two hours or more delay (irrespective of sleeping tablets).
(In either case, ten or more nights during month.)

** Do you seem to be slowed down in your movements, or to have too little energy recently? How much has it affected you?
(Do things seem to be moving too fast for you?)
RATE SUBJECTIVE ANERGIA AND RETARDATION. ☐ (36)
1 = Marked subjective listlessness and lack of energy.
2 = Marked retardation and underactivity (Irrespective of time during month).

IF NO APPETITE OR SLEEP DISTURBANCE, AND NO DEPRESSION, CUT OFF → SECTION 8.

Cut off ☐

IF SLEEP DISTURBANCE OR DEPRESSION:

Do you wake early in the morning?
RATE EARLY WAKING *(one hour before usual).* ☐ (37)
1 = One hour or more before ordinary time.
2 = Two hours or more before ordinary time.
(In either case, ten or more nights during month.)

Has there been any change in your interest in sex?
RATE LOSS OF LIBIDO WITHIN PRESENT EPISODE OF ILLNESS AND PERSISTING DURING PAST MONTH. ☐ (38)
1 = Marked loss of interest and performance.
2 = Almost total loss of libido.

Does the depression or tension get worst just before the start of the monthly period?
RATE PREMENSTRUAL EXACERBATION ☐ (39)
0 = No definite exacerbation.
1 = Marked exacerbation.

8. IRRITABILITY

** Have you been very much more irritable than usual recently?
(How do you show it?)
(Do you keep it to yourself, or shout, or even hit people?)
RATE IRRITABILITY. ☐ (40)
1 = Keeps irritation to himself.
2 = Shows anger by shouting or quarrelling.
3 = Shows anger by hitting people, throwing or breaking things.

9. EXPANSIVE MOOD AND IDEATION

** Have you sometimes felt particularly cheerful and on top of the world, without any reason?
(Too cheerful to be healthy?)
(How long does it last?)
RATE EXPANSIVE MOOD: *not ordinary high spirits.* ☐ (41)

1 = Moderately expansive mood (euphoria with marked element of inappropriateness or excitement, whether recognised by subject or not), present during past month, and persistent for hours at a time.* *Do not include transient high spirits.* Not necessarily described by subject.
2 = Intense form of symptom (elation or exaltation) definitely present during past month and persistent for hours at a time. Described by subject.

** Have you felt particularly full of energy lately, or full of exciting ideas?
(Do things seem to go too slowly for you?)
(Do you need less sleep than usual?)
(Do you find yourself extremely active but not getting tired?)
(Have you developed new interests recently?)
RATE SUBJECTIVE IDEOMOTOR PRESSURE. ☐ (42)

1 = Subjective equivalent of flight of ideas. Images and ideas flash through the mind, each suggesting others, at a faster rate than usual. State persists for hours at a time.* Definitely occurred during past month.
2 = As (1) but accompanied by very high energy output and activity which does not seem to make subject tired at the time. Definitely occurred during past month and persisted for hours at a time.*

IF NO EVIDENCE OF EXPANSIVE MOOD AND IDEATION, CUT OFF → SECTION 10.

Cut off ☐

IF EVIDENCE OF EXPANSIVE MOOD AND IDEATION:

Have you seemed super-efficient at work, or as though you had special powers or talents quite out of the ordinary?
Have you felt specially healthy?
Have you been buying any interesting things recently?
RATE GRANDIOSE IDEAS AND ACTIONS. ☐ (43)

1 = Subjective feeling of superb health, exceptionally high intelligence, extraordinary abilities, etc. Persistent for hours at a time.* Symptom occurred at some time during the month.
2 = Grandiose ideas have been translated into action during the month, e.g. overspending, gambling, etc., under the influence of grandiose ideas and expansive affect. *Do not include compulsive gambling unless clearly of this type.*

(→ GRANDIOSE DELUSIONS, SECTION 15D IF NECESSARY.)

* *If symptom was more transient but very intense or frequently repeated, it may still be included.*

10. OBSESSIONS

These symptoms are usually experienced as occurring against conscious resistance (see definition in glossary).

** Do you find that you have to keep on checking things that you know you have already done?
(Like gas taps, doors, switches, etc.)
(Do you have to touch or count things many times or repeat the same action over and over again?)
(What happens when you try to stop?)
RATE OBSESSIONAL CHECKING AND REPEATING. ☐ (44)

1 = Symptom of moderate intensity or, if severe, present less than 50% of the time.
2 = Symptom present in severe degree, more than 50% of the past month.

** Do you spend a lot of time on personal cleanliness, like washing over and over even though you know you are clean? What about tidiness?
(Do you get worried by contamination with germs?)
(Do you have other rituals?)
(What happens when you try to stop?)
RATE OBSESSIONAL CLEANLINESS AND SIMILAR RITUALS. ☐ (45)

1 = Symptom of moderate intensity or, if severe, present less than 50% of the time.
2 = Symptom present in severe degree, more than 50% of the past month.

** Do you find it difficult to make decisions even about trivial things?
(Do you constantly have to question the meaning of the universe?)
(Do you get awful thoughts coming into your mind even when you try to keep them out?)
(What happens when you try to stop?)
RATE OBSESSIONAL IDEAS AND RUMINATION. ☐ (46)

1 = Symptom of moderate intensity or, if severe, present less than 50% of the time.
2 = Symptom present in severe degree, more than 50% of the past month.

11. DEREALISATION AND DEPERSONALISATION

** Have you had the feeling recently that things around you were unreal?
(As though everything was an imitation of reality, like a stage set, with people acting instead of being themselves?)
(What is it like? How do you explain it?)
RATE DEREALISATION. ☐ (47)

1 = Moderately intense form of symptom definitely occurred during the past month, and persisted for hours at a time. Things appear colourless and artificial, people appear lifeless and seem to act rather than being themselves.

2 = Intense form of symptom occurred during the past month and persisted for hours at a time, e.g. whole world appears like a gigantic stage set, with imitation instead of real objects and puppets instead of people. (If delusional, do not rate here but symptom 90.)

** Have you yourself felt unreal, that you were not a person, not in the living world?
(Or that you were outside yourself, looking at yourself from outside?)
(Or that you look unreal in the mirror?)
(Or that some part of your body did not belong to you?)
(How do you explain it?)
RATE DEPERSONALISATION ☐ (48)

1 = Moderately intense form of the symptom definitely occurred during the past month and persisted for hours at a time. Subject feels himself unreal, a sham, a shadow.
2 = Intense form of symptom definitely occurred during the past month and persisted for hours at a time. Subject feels he is dead, not a person, living in a parallel existence, a hollow shell, even that he does not exist. (If delusional, do not rate here but symptom 90.)

12. OTHER PERCEPTUAL DISORDERS (NOT HALLUCINATIONS)

** Do you ever get the feeling that something odd is going on which you can't explain?
(Or that familiar surroundings seem strange? How do you explain it?)
RATE DELUSIONAL MOOD: *The subject feels that his familiar environment has changed in a way which puzzles him and which he may not be able to describe clearly. The feeling often accompanies delusion formation.* ☐ (49)

1 = Symptom definitely present. No delusions have actually been formulated, though patient may feel that various delusional explanations are possible.
2 = Full delusional elaboration has occurred.

** Does your imagination sometimes play tricks on you?

** Is there anything unusual about the way things look or sound, or smell, or taste?
(Does your body function normally?)
(Is your own appearance normal?)

CONTINUE BELOW CUT-OFF IF NECESSARY, EVEN IF (49) NOT PRESENT.

IF NO PERCEPTUAL ABNORMALITY → SYMPTOM 54.

Cut off ☐

IF THERE IS ANY HINT OF PERCEPTUAL ABNORMALITY, CONTINUE BEYOND CUT-OFF POINT AND ALSO CONSIDER LATER SECTIONS. RATE ONLY BASIC EXPERIENCE, NOT DELUSIONAL ELABORATION.

In what way? Do sounds seem unnaturally clear or loud, or things look vividly coloured or detailed?
(How do you explain this?)
RATE HEIGHTENED PERCEPTION: *e.g. subject intensely aware of cracks in a wall, details of a wallpaper pattern, colours in a picture. Sounds heard with exceptional clarity, music appears particularly beautiful.* ☐ (50)

1 = Subject unable to describe the symptom precisely, but examiner thinks it is likely to have been present at some time during the past month.
2 = Subject describes symptom. Definitely present at some time (even if only briefly) during the past month.

Do things seem dark or grey or colourless?
(How do you explain it?)
RATE DULLED PERCEPTION: *The reverse of symptom (50). Things look, sound and taste dull, flat, colourless and uninteresting.* ☐ (51)

1 = Subject unable to describe the symptom precisely, but examiner thinks it is likely to have been present at some time during the past month.
2 = Subject describes symptom. Definitely present at some time (even if only briefly) during the past month.

Does the appearance of things or people change in a puzzling way: e.g. distorted shapes or size or colour?
(How do you explain it?)
RATE CHANGED PERCEPTION. ☐ (52)

1 = Subject unable to describe the symptom precisely, but examiner thinks it is likely to have been present at some time during the past month.
2 = Subject describes symptom. Definitely present at some time (even if only briefly) during the past month.

Do you think your own appearance is normal?
(Conviction that nose is too large, teeth misshapen, body crooked, etc. Ask questions here if convenient but rate symptom (89).)

Does your experience of time seem to have changed?
(Does it go too fast or too slowly, or do you seem to live through experiences exactly as you have had them before?)
RATE CHANGED PERCEPTION OF TIME, INCLUDING *DEJA VU.* ☐ (53)

1 = Subject unable to describe the symptom precisely, but examiner thinks it is likely to have been present at some time during the past month.
2 = Subject describes symptom. Definitely present at some time (even if only briefly) during the past month.

Do you feel you have lost your emotions in some way?
(That you are empty of all feeling, incapable of reacting emotionally?)
(Is this a definite change, or have you always been like that?)
(How do you explain it?)

RATE LOST EMOTIONS: *Rate only subjective loss of affect, i.e. subject can remember being able to react emotionally, though this might have been months or even years ago.* ☐ (54)

1 = Symptom definitely present during the past month but less than 50% of the time.
2 = Symptom present more than 50% during the past month.

13. THOUGHT READING, INSERTION, ECHO, BROADCAST

IF QUESTION HAS NOT BEEN COVERED IN SECTION 4 ASK:

** Can you think quite clearly or is there any interference with your thoughts?
(Are you in full control of your thoughts?)
(Can people read your mind?)
(Is anything like hypnotism or telepathy going on?)

IF NO EVIDENCE OF THOUGHT READING, etc., CUT OFF → SECTION 14.

Cut off ☐

IF ANY EVIDENCE, ASK QUESTIONS BELOW:

(These symptoms are often recorded as false positives. The examiner must be satisfied that the subject is not simply assenting to a question he does not understand, but genuinely recognises the experience and can describe it so that the examiner recognises it.) It is particularly important to know the relevant sections of the Instruction Manual well before rating these symptoms.

Are thoughts put into your head which you know are not your own?
(How do you know they are not your own?)
(Where do they come from?)

RATE THOUGHT INSERTION: *Include only thoughts recognised as alien. Do not include delusional elaboration, only basic experience. (Exclude hallucinations,)* ☐ (55)

1 = Symptom described clearly, but subject thinks it may be due to 'own unconscious thoughts' etc., i.e. not certainly alien.
2 = Symptom described clearly and thoughts described as alien, i.e. inserted into mind from elsewhere (even if subject does not know from where). Not hallucinations.

Do you ever seem to hear your own thoughts spoken aloud in your head, so that someone standing near might be able to hear them?
(Are your thoughts broadcast, so that other people know what you are thinking?)
(How do you explain it?)

RATE THOUGHT BROADCAST. ☐ (56)

1 = Hears own thoughts 'spoken' aloud but not broadcast. Subject must really hear them aloud in his head. If in doubt rate (8) or (0).
2 = Thoughts transferred or broadcast so that others can share subject's thoughts even when they are not in the same room. (Do not include 'thoughts being read' unless this is an explanation of thought broadcast. The subject must actually experience his thoughts being available to others.)

Do you ever seem to hear your own thoughts repeated or echoed?
(What is that like? How do you explain it?)
(Where does it come from?)

RATE THOUGHT ECHO OR COMMENTARY. ☐ (57)

1 = Thought echo. If any doubt, rate (8) or (0).
2 = Subject experiences alien thoughts related to his own thoughts, i.e. associations or comments on his own thoughts. Not hallucinations.

Do you ever experience your thoughts stopping quite unexpectedly so that there are none left in your mind, even when your thoughts were flowing freely before?
(What is that like?)
(How often does it occur? What is it due to?)

Do your thoughts ever seem to be taken out of your head, as though some external person or force were removing them?
(Can you give an example?)
(How do you explain it?)

RATE THOUGHT BLOCK OR WITHDRAWAL. ☐ (58)

1 = Thought block. Do not include if due to anxiety or lack of concentration; only if it occurs totally unexpectedly when thoughts are flowing freely. One single occasion is not sufficient for rating. *Be very critical in rating this symptom.*
2 = Delusional explanation that thoughts are withdrawn.

Can anyone read your thoughts?
(How do you know? How do you explain it?)

RATE DELUSION OF THOUGHTS BEING READ: *Only if subject does not mean that people can infer his thoughts from his actions. (Do not include subject reading thoughts of other people → 76.)* ☐ (59)

1 = 'Partial' delusion. Subject entertains the possibility that thoughts might be read but is not certain about it. Exclude if subcultural explanation.
2 = Full delusion. Exclude if subcultural explanation. The term 'thought reading' is commonly used to mean the ability to tell what someone is thinking from the way they behave – this use should be excluded.

14. HALLUCINATIONS

USE JUDGEMENT ABOUT WORDING.

** I should like to ask you a routine question which we ask of everybody. Do you ever seem to hear noises or voices when there is no one about, and nothing else to explain it?
(Do you ever seem to hear your name being called?)

** Is that true of visions or other unusual experiences, which some people have? (Touch, taste, smell, temperature, pain, etc.)

IF NO EVIDENCE FOR HALLUCINATIONS OF ANY SENSE, CUT OFF → SECTION 15.

Cut off ☐

IF EVIDENCE FOR NON-AUDITORY HALLUCINATIONS ONLY → SUBSECTIONS 14B and 14C

14A. AUDITORY HALLUCINATIONS

IF ANY EVIDENCE THAT AUDITORY HALLUCINATIONS MIGHT BE PRESENT:

Do you hear noises like tapping, or music? (What is it like?)
Does it sound like muttering or whispering?
Can you make out the words?
RATE NON-VERBAL AUDITORY HALLUCINATIONS. ☐ (60)

1 = Music, tapping, car engines, etc. Do not include tinnitus.
2 = Muttering, whispering but subject cannot make out any words at all.

What does the voice say?
(Write down examples of typical verbal hallucinations.)
(If accusatory: Do you think that it is justified? Do you deserve it?)
Do you hear your name being called?
RATE VERBAL HALLUCINATIONS BASED ON DEPRESSION OR ELATION OR VOICE CALLING SUBJECT. ☐ (61)

Content is congruent with mood; e.g. 'He's dirty', in context of depression, or 'Go to Westminster', in elated subject who thinks he is Prime Minister. Include voice calling subject (e.g. calling name) or saying single words only. Be careful to distinguish from delusions of reference in which people whom the subject can see are thought to be talking about him.

RECORD EXAMPLES.

1 = Voice calling name, or single words only.
2 = Other verbal hallucinations; congruent with depressed mood.
3 = Other verbal hallucinations; congruent with elated mood.

Do you hear several voices talking about you?
Do they refer to you as 'he' (she)?
(What do they say?)
(Do they seem to comment on what you are thinking, or reading, or doing?)

RATE VOICE(S) DISCUSSING SUBJECT IN THIRD PERSON OR COMMENTING ON THOUGHTS OR ACTIONS (NOT BASED ON DEPRESSION OR ELATION). ☐ (62)

Do not include muttering or whispering if subject cannot make out words. Exclude 'dissociative' hallucinations (symptom 64). Do not include voice calling name or affectively based verbal hallucinations (symptom 61). There may be one voice commenting on subject's thoughts or actions, or several voices discussing the subject in the third person.

RECORD EXAMPLES.

1 = Hears a voice or voices commenting on thoughts or actions in third person (e.g. 'Now he's going to go to bed' or 'Why would he think a thing like that?'). (2) not present.
2 = Hears voices talking about him/her in third person (e.g. 'I think he's a homosexual, don't you?' 'Yes, he wears a pink pullover, that's a sign of it.'). (1) may also be present.

Do they speak directly to you?
(Are they threatening or unpleasant?)
(Do they call you names?)
Do they give orders? (Do you obey?)

RATE VOICE(S) SPEAKING TO SUBJECT (NOT BASED ON DEPRESSION OR ELATION). ☐ (63)

Include voice(s) speaking directly to subject, whether accusing, threatening, giving orders or giving information. Exclude voice(s) calling name or based on depression or elation (symptom 61), or commenting on subject's thoughts or actions (symptom 62). Exclude 'dissociative' hallucinations (symptom 63).

RECORD EXAMPLES.

1 = Pleasant, supportive or neutral voice(s), not based on affect. No hostile voices.
2 = Hostile, threatening or accusing voice(s), thought to be undeserved and not based on affect.

N.B. If single isolated words, even with neutral affect, include under 61 (1).

Can you carry on a two-way conversation with —?
(You can reply, and then — replies to you, and you reply again, just as in an ordinary conversation?)
(Do you see anything, or smell anything at the same time as you hear the voice?)
(Who is it you are talking to?)
(What is the explanation?)
(Do you know anyone else who has this kind of experience?)

RATE 'DISSOCIATIVE' HALLUCINATIONS (VERBAL AND/OR OTHER) ☐ (64)

The subject can hold a two-way conversation with a presence (variously described as a person, ghost, spirit, god, etc.) which may also be sensed in other ways, e.g. visually or by touch or smell. Often connected with people with whom the subject has had strong affective ties. Visual hallucinations can occur alone. There is usually a strong subcultural colouring, e.g. the subject belongs to a religious sect or to a subcultural group which sanctions hallucinatory experiences, or the subject has been under the influence of someone who is involved with such practices. Exclude hypnogogic hallucinations.

RECORD EXAMPLES.

1 = 'Dissociative' hallucinations present. Subject belongs to subcultural group or sect in which such experiences are sanctioned.
2 = 'Dissociative' hallucinations present. Subject does not belong to subcultural group as in (1). If not known, rate (1).

Are these voices in your mind or can you hear them through your ears?

Scoring: ☐ (65)

1 = Subject hears both pseudo-hallucinations (within mind) and true hallucinations (through ears).
2 = Subject hears pseudo-hallucinations only.
3 = Subject hears true hallucinations only.

How do you explain the voice?

RECORD EXPLANATION.

14B. VISUAL HALLUCINATIONS

IF QUESTION HAS NOT BEEN COVERED IN SECTION 12 OR 14A, ASK:

** Have you had visions, or seen things other people couldn't see?

IF NO EVIDENCE, HERE OR ELSEWHERE, FOR VISUAL HALLUCINATIONS CUT OFF → SECTION 15.

Cut off ☐

IF ANY EVIDENCE OF VISUAL HALLUCINATIONS:

With your eyes or in your mind?
What did you see?
Were you half asleep at the time?
Has it occurred when you were fully awake?
Did you realise you were 'seeing things'?
Did the vision seem to arise out of a pattern on the wallpaper or a shadow?
How do you explain it?

RATE VISUAL HALLUCINATIONS: *in clear consciousness including pseudo-hallucinations. Exclude 'dissociative' visual hallucinations (symptom 64).* ☐ (66)

1 = Formless visual hallucinations – flashes of light, shadows, etc.
2 = Formed visual hallucinations – people, objects like a 'fiery cross', faces, etc.

RATE DELIRIOUS VISUAL HALLUCINATIONS. ☐ (67)

14C. OTHER HALLUCINATIONS

IF QUESTIONS HAVE NOT BEEN COVERED IN PREVIOUS SECTIONS:

** Is there anything unusual about the way things feel, or taste, or smell?

** Does your body function normally?

IF NO EVIDENCE FOR OTHER HALLUCINATIONS CUT OFF → SECTION 15A.

Cut off ☐

IF ANY EVIDENCE FOR OTHER HALLUCINATIONS:

Do you sometimes notice strange smells that other people don't notice?
(What sort of thing?)
(How do you explain it?)

RATE OLFACTORY HALLUCINATIONS: *Exclude delusion that patient himself smells.* ☐ (68)

1 = Simple olfactory hallucination. Not delusionally elaborated. Subject smells oranges, death, a burnt smell, scent, etc., which other people cannot smell. Can offer no explanation.
2 = Delusional elaboration in addition, e.g. gas being put into room.

Do you seem to think that you yourself give off a smell which is noticed?
(What is the explanation?)

RATE DELUSION THAT SUBJECT SMELLS: *Do not include simple preoccupation with body odour, e.g. in anxious subject who sweats a lot.* ☐ (69)

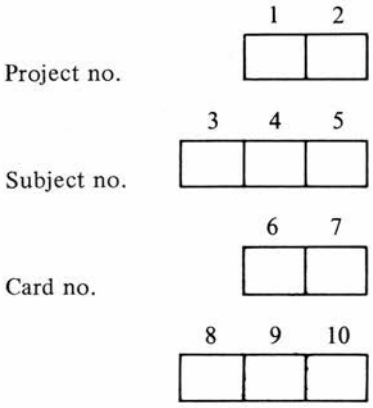
1 = Subject irrationally thinks he gives off a smell but is not certain. Not sure that others have noticed it but thinks it possible.
2 = Subject sure that he gives off a smell and that others have noticed it and react accordingly.

Do you ever feel that someone is touching you, but when you look there is nobody there?)
(Have you noticed that food or drink seems to have an unusual taste recently?)

RATE OTHER HALLUCINATIONS AND DELUSIONAL ELABORATION: *Exclude hypochondriacal and nihilistic delusions rated in (90) and (91).* ☐ (70)

1 = Sensation of touch, food tastes burnt, etc., but subject puzzled by the experience. No delusional elaboration.

2 = Delusional elaboration in addition, e.g. fantasy lover, food poisoned, etc.



15. DELUSIONS

Definition

Delusions may be of two kinds, primary and secondary. Both kinds are rated together in the following symptoms except where specified. For example, primary delusions are specifically rated in symptom (82). They are defined here for convenience.

Primary delusions are based upon experiences in which a subject suddenly becomes convinced that a particular set of events has a special meaning (e.g. a subject undergoing a liver biopsy suddenly felt he had been chosen by God). The delusion cannot be explained and it is not shared by other members of the subject's cultural or social group.

Secondary delusions are delusional elaborations of primary delusions or other basic phenomena such as derealisation, depersonalisation, perceptual distortions, hallucinations, thought echo, mood changes, etc.

Above cut-off questions, likely to elicit delusions if present, are included in many of the preceding sections. There may also be evidence in the case-record or in the subject's spontaneous account.

IF NO EVIDENCE AT ALL THAT DELUSIONS ARE PRESENT, CUT OFF → SECTION 16.

RECORD IF ANY PSYCHOTIC PHENOMENA PRESENT, OTHER THAN DELUSIONS, USE JUDGEMENT AS TO WHETHER TO PROCEED BEYOND CUT-OFF.

Cut off

IF ANY EVIDENCE FOR DELUSIONS, ASK ALL QUESTIONS NOT IN BRACKETS, AND ANY FURTHER QUESTIONS WHICH SEEM INDICATED.

RATING OF PARTIAL AND FULL DELUSIONS.

In general, all delusions are rated as follows:

1 = Partial delusions, which are expressed with doubt, or as possibilities which the subject entertains but is not certain about. This rating should not be used if it is clear that full delusions have been present during the month, or if the subject has acted as if fully deluded.

2 = Full delusions have been present at some time during the month. Fully convinced. No insight.

A useful question to elucidate the difference between partial and full delusions is as follows:

Even when you seem to be most convinced, do you really feel in the back of your mind that it might well not be true, that it might be imagination?

15A. DELUSIONS OF CONTROL

Definition

The subject's will is replaced by that of some external agency. A simple statement that the radio is controlling the subject is not sufficient. (This statement, alone, should be rated 8.) The subject must describe a replacement of will by some other force.

Do not include feeling that life is planned and directed by fate, or that the future is present already in embryo, or that subject is not very strong-willed, or that voices give subject orders. Do not include simple identification with God or being under God's direction. Do not include subcultural or hysterical possession states or multiple personality (→ 100).

Do you feel under the control of some force or power other than yourself?
(As though you were a robot or a zombie without a will of your own?)
(As though you were possessed by someone or something else?)
(What is that like?)
(Does this force make your movements for you without your willing it, or use your voice, or your handwriting? Does it replace your personality? What is the explanation?)

RATE DELUSIONS OF CONTROL. ☐ (71)

1 = Partial delusions 2 = Full delusions

15B. MISINTERPRETATIONS, MISIDENTIFICATION AND DELUSIONS OF REFERENCE

Definition

Delusions of reference: Do not include simple self-consciousness or feeling that subject attracts comment, even if critical. These are rated under symptom 31.

There must be elaboration: e.g. someone crosses his knees in order to indicate that the subject is homosexual; or the whole neighbourhood is gossiping.

Delusional misinterpretations, etc. This is an extension of the delusion of reference, so that not only do people seem to refer to subject, but situations appear to be deliberately created to test him (exclude situations of medical treatment), or objects appear to have special meanings.

Do people seem to drop hints about you or say things with a double meaning, or do things in a special way so as to convey a meaning?
Does everyone seem to gossip about you?
(Do people follow you about or check up on you or record your movements?)
(How do they do it? Why?)
(Are there people about who are not what they seem to be?)
RATE DELUSIONS OF REFERENCE. ☐ (72)

1 = Partial delusions 2 = Full delusions.

Do things seem to be specially arranged?
(Is an experiment going on, to test you out?)
(Do you see any reference to yourself on TV or in the papers?)
(Do you ever seem to see special meanings in advertisements, or shop windows, or in the way things are arranged?)
(How do you explain this?)
RATE DELUSIONAL MISINTERPRETATION AND MISIDENTIFICATION. ☐ (73)

1 = Partial delusions 2 = Full delusions

15C. DELUSIONS OF PERSECUTION

Is anyone deliberately trying to harm you, e.g. trying to poison you or kill you?
(How? Is there an organisation like the Mafia behind it?)
(Is there any other kind of persecution? How do you explain this?)
RATE DELUSIONS OF PERSECUTION. ☐ (74)

1 = Partial delusions 2 = Full delusions

15D. EXPANSIVE DELUSIONS

Do you think that people are organising things specially to help you?
RATE DELUSIONS OF ASSISTANCE. ☐ (75)

1 = Partial delusions 2 = Full delusions

Is there anything special about you? Do you have special abilities or powers?
(Can you read people's thoughts?)
(Is there a special purpose or mission to your life?)
(Are you especially clever or inventive? How do you explain this?)
RATE DELUSIONS OF GRANDIOSE ABILITIES. ☐ (76)

1 = Partial delusions 2 = Full delusions

(Are you a very prominent person or related to someone prominent, like Royalty?)
(Are you very rich or famous?)
(How do you explain this?)

RATE DELUSIONS OF GRANDIOSE IDENTITY: *(Exclude religious identification.)* ☐ (77)

1 = Partial delusions 2 = Full delusions

15E. DELUSIONS CONCERNING VARIOUS TYPES OF INFLUENCE AND PRIMARY DELUSIONS

Are you a very religious person?
(Specially close to Christ or God?)
(Can God communicate with you? How?)
(Are you yourself a saint?)
(How do you explain this?)

RATE RELIGIOUS DELUSIONS: *Including delusional religious explanations of other experiences. Exclude intense religious belief or purely subcultural beliefs.* ☐ (78)

1 = Partial delusions 2 = Full delusions

How do you explain the things that have been happening? (SPECIFY)
Is there anything like hypnotism, telepathy, or the occult going on?
What is the explanation?

INCLUDE DELUSIONAL EXPLANATIONS IN TERMS OF PARANORMAL PHENOMENA: *e.g. hypnotism, telepathy, magic, witchcraft, etc. Exclude purely subcultural beliefs, → 83.* ☐ (79)

1 = Partial delusions 2 = Full delusions

Is anything like electricity, or X-rays, or radio-waves affecting you?
(In what way? What is the explanation?)

INCLUDE DELUSIONAL EXPLANATIONS IN TERMS OF PHYSICAL FORCES: *e.g. radio, television, X-rays, electricity, transmitters, microphones, machines of various kinds.* ☐ (80)

1 = Partial delusions 2 = Full delusions

DELUSIONS OF ALIEN FORCES PENETRATING OR CONTROLLING MIND (OR BODY). ☐ (81)

Include any delusion, whether rated elsewhere or not, which involves an external force penetrating the subject's mind or body, e.g. rays turn liver to gold, alien thoughts pierce skull or are inserted into mind, hypnotism makes patient levitate, a spirit speaks with subject's voice, a radio transmitter has been implanted into brain and broadcasts thoughts or controls actions, etc.

1 = Partial delusions 2 = Full delusions

Choose a likely delusion, and ask:

How did it come into your mind that this was the explanation?
(Did it happen suddenly? How did it begin?)

RATE PRIMARY DELUSIONS: *Based upon experiences in which subject suddenly becomes convinced that a particular set of events has a special meaning. (See definition on page 214.) Not based on mood or explanation of other abnormal experiences.* ☐ (82)

1 = Partial delusions 2 = Full delusions

15F. OTHER DELUSIONS

(Examiner should question as appropriate.)

RATE SUBCULTURALLY INFLUENCED DELUSIONS: *Include only subjects who belong to small groups with definitely idiosyncratic beliefs; small sects, tribes, 'secret societies', etc.* ☐ (83)

0 = No significant subcultural influence. For example, an English subject believing he is influenced by TV would be rated (0) since, although the delusion depends on TV being available in England, it is not in any way specific to a small subcultural group.

1 = One or more of the 'delusions' rated earlier could easily be no more than a belief shared by other members of the subject's subcultural group, e.g. the Pentecostal church with the gift of tongues. Voodoo, witchcraft, communicating with God, are other examples of beliefs which may be taken quite literally by groups of people who are not clinically deluded. Rate (1) if subject holds such beliefs without elaborating them further.

2 = As (1), but because of excitement, expansiveness, depression, confusion, intellectual retardation, etc., the subject holds the beliefs with exceptional fervour and conviction, or elaborates them further. Such a subject might well be regarded as abnormal by other members of his own sect or group.

3 = More specific delusional states, e.g. Koro, Witigo, etc.

(Do you have any reason to be jealous of anybody?)

MORBID JEALOUSY. ☐ (84)

1 = Partial delusions 2 = Full delusions

DELUSION OF PREGNANCY. ☐ (85)

1 = Partial delusions 2 = Full delusions

SEXUAL DELUSIONS: *Any delusion with sexual content, e.g. fantasy lover, sex changing, etc. Do not include an untrue claim that a subject is married or has children.* ☐ (86)

1 = Partial delusions 2 = Full delusions

Have you had any unusual experience or adventures recently?
RATE FANTASTIC DELUSIONS, DELUSIONAL MEMORIES, DELUSIONAL CONFABULATIONS, FANTASTIC DELUSIONS:
Confabulation: Subject makes up delusions on the spot. Very rare.
Delusional memories: Subject seems to be describing actual memories. Describes the same delusions time and again. Not confabulations. Rare, e.g. 'I came down to earth on a silver star.' Fantastic delusions: The commonest of the three, e.g. England's coast melting. ☐ (87)

1 = Partial delusions 2 = Full delusions

15G. SIMPLE DELUSIONS BASED ON GUILT, DEPERSONALISATION, HYPOCHONDRIASIS, ETC.

Definition

These symptoms often appear to be based on a depressed mood and are relatively consistent and unelaborated. Do not include more bizarre elaborations of any of them, e.g. having a metal nose = symptom 87, not 89. Having been turned into another specified person = possibly symptom 71, not 90. Liver turned to lead by X-rays = symptoms 80 and 81, not 91. England's coast melting = symptom 87, not 92.

Do you feel you have committed a crime, or sinned greatly, or deserve punishment? (Have you felt that your presence might contaminate or ruin other people?)
RATE DELUSIONS OF GUILT. ☐ (88)

1 = Subject has brought ruin to family by being in present condition, or thinks that symptoms are a punishment for not doing better, etc. Does not elaborate as in (2).
2 = Subject says has sinned greatly or committed some terrible crime or brought ruin upon the world. May feel deserving of punishment, even of death or hell-fire, because of it.

(Do you think your appearance is normal?)
RATE SIMPLE DELUSIONS CONCERNING APPEARANCE: *(Nose too large, teeth misshapen, body crooked, etc.)* ☐ (89)

1 = Strong feeling that there is something wrong with appearance; subject looks old or ugly or dead, skin cracked, teeth misshapen, nose too large, body crooked, etc. Can be reassured temporarily. There may be only one limited preoccupation.
2 = Subject acts accordingly (plastic operations, etc.)

(Is anything the matter with your brain?)
RATE DELUSIONS OF DEPERSONALISATION: *Subject has no head, does not exist, hollow instead of a brain, etc.* ☐ (90)

1 = Unable to think, no thoughts in head, feels *as though* he has no brain or as though it does not function at all.
2 = Symptom more intense. Subject has no head, no brain, does not exist.

(Is anything the matter with your body?)

RATE HYPOCHONDRIACAL DELUSIONS: *Subject has incurable cancer, bowels are stopped up, insides are rotting, etc.* ☐ (91)

1 = Subject feels body is unhealthy, rotten, diseased, but without the force of (2).
2 = Subject has incurable cancer, bowels are stopped up or rotting away, etc.

(Do you have the feeling that something terrible is going to happen? What?)

RATE DELUSIONS OF CATASTROPHE: *World is about to end, some catastrophe has happened or will occur, everything is evil and will be destroyed.* ☐ (92)

1 = Subject feels sense of impending doom; something awful will happen. Non-specific but out of proportion to circumstances.
2 = Delusional conviction that world is about to end or some other enormous catastrophe is about to occur or has occurred. World is dirty, decayed, rotten: i.e. further delusional elaboration of (1).

15H. GENERAL RATINGS OF DELUSIONS AND HALLUCINATIONS

(Include both partial and full delusions.)

CONSIDER BOTH DELUSIONS AND HALLUCINATIONS IN FOLLOWING RATINGS.

RATE SYSTEMATISATION OF DELUSIONS. ☐ (93)

Scoring:
0 = No delusions or hallucinations.
1 = Delusions and hallucinations not elaborated into a general system affecting much of the subject's experience. Include encapsulated delusions or isolated hallucinations.
2 = Some systematic elaboration, but substantial areas of the subject's experiences are not affected.
3 = Subject interprets practically all his experience in delusional terms.

RATE EVASIVENESS. ☐ (94)

Scoring:
0 = No attempt at concealment suspected.
1 = Examiner suspects that there may be (either) delusions or hallucinations in the background, but the subject is not concealing much of the psychopathology.
2 = Examiner suspects that there is a considerable preoccupation with delusions (even a delusional system) or hallucinations, but the subject tries to conceal them.
3 = No concealment but other delusions or hallucinations probably present. Not elicited because of poor intelligence and education or incoherence or muteness, etc.

OVERALL RATING OF PREOCCUPATION WITH DELUSIONS AND HALLUCINATIONS. ☐ (95)

Scoring:

0 = No delusions or hallucinations.
1 = No delusions or hallucinations definitely rated but examiner suspects that they may be present.
2 = Preoccupied with past delusions or hallucinations only. Not actively deluded or hallucinated at present.
3 = Delusions or hallucinations definitely present but subject is not preoccupied with them for much of the time. Can turn attention to other things without difficulty.
4 = Delusions or hallucinations present and take up most of the subject's attention. Preoccupied to the exclusion of many other matters.
5 = Patient can hardly discuss anything but delusions.

RATE ACTING OUT DELUSIONS ☐ (96)
(Rate from case-record, etc.)

Scoring:

0 = No delusions or hallucinations.
1 = Subject able to keep delusions or hallucinations to himself, or to confide them only to a few trusted people (sympathetic relatives, friends, doctors, etc.). He does not express them in public nor act upon them. Does not talk out loud to voices.
2 = Subject has acted upon delusions or hallucinations during past month, or expressed them in public (i.e. outside the small circle of people who would be expected to be sympathetic). This has not, however, resulted in severe social disturbance or a social crisis.
3 = As (2) but acting out, or public expression, has resulted in severe social disturbance or a social crisis.

16. SENSORIUM AND FACTORS AFFECTING

** Have you had any lapses of memory recently?
(Have there been any periods in which you completely forgot what happened?)
(What was it like?)
(How do you explain it?)

RATE FUGUES, BLACKOUTS, AMNESIA LASTING MORE THAN ONE HOUR: *irrespective of aetiology.* ☐ (97)

1 = less than 12 hours.
2 = 12–24 hours.
3 = more than 24 hours.

** What medicines or drugs do you take?
(Do you take anything for your nerves or your mood?)
(Obtain list of drugs.)
(Who prescribes?)

RATE DRUG ABUSE DURING MONTH. *One category only.* (98)

1 = Cannabis.
2 = Amytal, etc.
3 = LSD, amphetamine, etc.
4 = Cocaine, heroin, etc.

** May I ask about your drinking habits? How much do you usually drink each day?
(Is alcohol in any way a problem for you? In what way?)

(CHECK LIST: *Present on card if needed.* During the past month have you:
had family problems because of drinking?
missed work because of drinking?
had morning shakes or other withdrawal symptoms?
had blackouts for several hours?
heard voices or seen visions?)

RATE ALCOHOL ABUSE DURING PAST MONTH. (99)

1 = Agrees alcohol has been a problem but not 2.
2 = Any check-list item applies.

RATE DISSOCIATIVE STATES DURING PAST MONTH:
'Narrowing of consciousness which serves an unconscious purpose and is commonly accompanied or followed by a selective amnesia', e.g. trance, possession state, fugue, hypersomnia, stupor, etc. Do not include if caused by drugs, alcohol, epilepsy, etc. (100)

1 = Present during the past month, but not at examination.
2 = Present at examination.

RATE CONVERSION SYMPTOMS, *e.g. paralysis, anaesthesia, blindness, tremor, seizures, etc. if mentioned during interview.* (101)

1 = Present during month, not at examination.
2 = Present at examination.

RATE CLOUDING OR STUPOR AT EXAMINATION (102)

1 = Clouding: Inadequate comprehension of external impressions, with perplexity, and impairment of attention and orientation.
2 = Stupor: Subject appears comatose but there is no clouding or impairment of consciousness.

IF ANY SUSPICION OF POOR MEMORY OR DISORIENTATION:

May I ask one or two standard questions we ask of everybody?
How old are you?
Can you tell me the year and the month?
What is the name of the Prime Minister?

RATE ORGANIC IMPAIRMENT OF MEMORY. *See glossary for definition.* ☐ (103)

1 = Mild.
2 = Moderate.
3 = Severe.

17. INSIGHT

** Do you think there is anything the matter with you?
(What do you think it is?)
(Could it be a nervous condition?)
(What do you think the cause is?)
(Why did you need to come to hospital?)
(Do you think (*specify delusions or hallucinations*) were part of a nervous condition?)

IF PSYCHOTIC SYMPTOMS (i.e. SYMPTOMS FROM SECTIONS 12–15): ☐ (104)

0 = Full insight (in intelligent subject, able to appreciate the issues involved).
1 = As much insight into the nature of the condition as social background and intelligence allow.
2 = Agrees to a nervous condition but examiner feels that subject does not really accept the explanation in terms of a nervous illness (e.g. gives delusional explanation, the result of persecution, or rays, etc.).
3 = Denies nervous condition entirely.
9 = Psychotic illness not present.

IF NEUROTIC SYMPTOMS (i.e. SYMPTOMS FROM SECTIONS 1–11 ONLY): ☐ (105)

0 = Full insight (in intelligent subject, able to appreciate the issues involved).
1 = As much insight into the nature of the condition as social background and intelligence allow.
2 = Gives physical explanation for neurotic symptoms.
3 = Denies neurotic symptoms entirely.
9 = Neurotic illness not present.

** Of all the problems you have told me about, which one affects you most?
How much does it interfere with your work or your relationships with other people?
(Have you actually been out of work, or been unable to do the housework, or go shopping, travelling, etc., during the past month?)
(Have the symptoms impaired your efficiency in any other way?)

RATE SOCIAL IMPAIRMENT DUE TO NEUROTIC CONDITION. ☐ (106)

0 = No neurotic or psychotic symptoms present.
1 = Neurotic symptoms present but little diminution of subject's efficiency or interference with everyday activities.

2 = Neurotic symptoms interfere with subject's efficiency to a moderate extent but are not incapacitating, e.g. subject neglects housework or can't enjoy leisure activities or social relationships, or finds work-efficiency reduced because of worry, tension, irritability, depression, anxiety, etc. Subject does not, however, stop work altogether or completely neglect household.
3 = Subject severely incapacitated by neurotic symptoms: had to have at least a week off work during past month; was housebound for a week or more; was actively withdrawn from all social relationships, etc. The subject does not have to be totally incapacitated for the whole month for this rating to be made, but impairment has to be very severe.
8 = Examiner unsure.
9 = Psychotic condition present.

(If both psychotic and neurotic condition, rate whichever shows more impairment.)

RATE SOCIAL IMPAIRMENT DUE TO PSYCHOTIC CONDITION ☐ (107)

0 = No neurotic or psychotic symptoms present.
1 = Psychotic symptoms present but little diminution of subject's efficiency or interference with everyday activities.
2 = Psychotic symptoms interfere with subject's efficiency to a moderate extent but are not incapacitating, e.g. subject neglects housework or can't enjoy leisure activities or social relationships, or finds work-efficiency reduced. Subject does not, however, stop work altogether or completely neglect household.
3 = Subject severely incapacitated by psychotic symptoms: had to have at least a week off work during past month; was housebound for a week or more; was actively withdrawn from all social relationships, etc. The subject does not have to be totally incapacitated for the whole month for this rating to be made, but impairment has to be very severe.
8 = Examiner unsure.
9 = Neurotic condition, and no psychotic condition, present.

FINAL QUESTION

** Have there been any other things lately that I haven't covered?
Specify;

Note here any points that seem to be important or unusual about the subject or the interview which are not covered in the schedule.

Reconsider schedule to make sure that all obligatory questions have been asked. Also consider whether behaviour, affect and speech ratings can be made or whether further observation or examination is necessary. IF NOT, THIS IS THE END OF THE INTERVIEW.

18–20. BEHAVIOUR, AFFECT AND SPEECH

RATINGS

0 = Symptom absent.
1 = Present in fairly severe degree, or very severe but intermittent during interview.
2 = Present in very severe degree and almost continuous during interview.
8 = Examiner not sure.
9 = Subject not examined, or examination not appropriate.

N.B. If in doubt, rate (0). A rating of (1) means there is no doubt about the symptom being present in fairly severe form.

Behaviour during interview

Self-neglect (cleanliness, shaven, make-up, state of hair and clothes). ☐ (108)

Bizarre appearance (secret documents openly displayed, special clothes or ornaments with symbolic significance, etc. Do not include mannerisms or posturing = symptom 116). ☐ (109)

Slowness and underactivity (sits abnormally still, walks abnormally slowly, delay in performing movements). ☐ (110)

Agitation (fidgety, restlessness, pacing, frequent unnecessary movements). ☐ (111)

Gross excitement and violence (throws things, runs or jumps about, waves arms wildly, shouts or screams). ☐ (112)

Irreverent behaviour (sings, facetious, silly jokes, flippant remarks, unduly familiar). ☐ (113)

Distractibility (stops talking or changes subject due to distraction by trivial noises or events outside the room or turns attention to furniture, etc.). ☐ (114)

Embarrassing behaviour (making sexual suggestions or advances to interviewer; loss of social restraint – scratches genitals, passes loud flatus, etc.). ☐ (115)

Mannerisms and posturing (odd, stylised movements or acts, usually idiosyncratic to the patient, often suggestive of special meaning or purpose: assuming and maintaining uncomfortable or inappropriate postures). ☐ (116)

Stereotypies, etc. (constant repetition of movements or postures such as rocking, rubbing, nodding, grimacing: no special significance). ☐ (117)

Behaves as if hallucinated (non-verbal: as though hears voices or visions: lips move soundlessly, looks round, giggles to self – not just from embarrassment, shyness, etc.). ☐ (118)

Catatonic movements (119)

(Negativism: does the opposite of what he is asked.
Ambitendence: begins to take proffered hand, then withdraws; etc.
Echopraxia: imitates examiner's movement.
Flexibilitas cerea: arm remains where it is put, for at least 15 seconds.
Mitgehen: excessive co-operation in passive movement.
Echolalia: imitates words and phrases with same intonation and inflection of voice.)
(These items can be separately rated in special projects.)

Affect during interview

Observed anxiety (tense worried look or posture, fearful apprehensive look, frightened tone of voice, tremor). (120)

Observed depression (sad, mournful look, tears, gloomy tone of voice, deep sighing, voice chokes on distressing topic). (121)

Histrionic (feelings expressed in exaggerated, dramatic, histrionic manner). (122)

Hypomanic affect (unduly cheerful, smiling, euphoric, elated). (123)

Hostile irritability (unco-operative, irritable, angry, overtly hostile, discontented, haughty, antagonistic). (124)

Suspicion. (125)

Perplexity (puzzlement). (126)

Lability of mood (whether lability of one mood, or changing from one mood to another). (127)

Blunted affect (expressionless face and voice, uniform blunting whatever the topic of conversation, indifference to distressing topics, whether delusional or normal). (128)

1 = Blunting not uniform, e.g. at times responds affectively but at other times is markedly flat; or responds with some evidence of affect, but definitely less than expected.
2 = Severe and uniform blunting.

Incongruity of affect (emotion is shown, but not congruent with topic). (129)

Speech during interview

Slow speech (long pauses before answering, long pauses between words). (130)

Pressure of speech (more copious speech than normal, too rapid speech, very loud voice, too circumstantial speech). (131)

Non-social speech (talks, mutters, whispers out loud, out of context of conversation with examiner). (132)

Muteness (133)

1 = Almost mute, fewer than twenty words in all.
2 = Totally mute.

Restricted quantity of speech (subject frequently fails to answer, questions have to be repeated, restricted to minimum necessary, no extra sentences, no additional comments). (134)

Neologisms and idosyncratic use of words or phrases, e.g. 'One is called "Per-God" and the other is called "Per-the-Devil"', '... miracle-willed through God's "tarn-harn"...', 'Well, there is a frequenting of clairvoyance...': 'Per-God', 'Per-the-Devil' and 'tarn-harn' are neologisms; 'frequenting of clairvoyance' is an example of ordinary words used idiosyncratically. DO NOT RATE THIS SYMPTOM PRESENT UNLESS EXAMPLES ARE WRITTEN DOWN. (135)

Disorder of content of speech

Three types of disordered content are specified: in each case, the effect is to make it very difficult to grasp what the subject means. However, the symptoms are defined in terms of specific components so that it should, in most cases, be possible to say whether one, two, or all three symptoms are present. If in doubt, rate hierarchically, i.e. rate incoherence in preference to flight of ideas and flight of ideas in preference to poverty of speech.

If the patient does not talk enough to give a rateable sample of speech, rate all three symptoms Y.

Incoherence of speech. The subject's meaning is obscured by distorted grammar, lack of logical connection between one part of a sentence and another or between sentences, sudden irrelevances or 'Knight's move', grossly pedantic phrases, answering off the point, etc. For example: (136)

'We've seen the downfall of the radium crown by the Roman Catholics, whereas when you come to see the drinking side of the business, God saw that Noah, if he lost his reason, he got nobody there to look after them.'

'I did suggest to you, that intrinsic or congenital sentiment or refinement of disposition would be so miracle-willed through God's "tarn-harn" as to assume quite the opposite.'

'I believe we live in a world, in an age, where the elements are a force that elders of professionalism hope, not to conquer, but to control.'

'What's your address?' 'It's supposed to be Salisbury near Birmingham.' (*Vorbeireden.*)

DO NOT RATE THIS SYMPTOM PRESENT UNLESS EXAMPLES ARE WRITTEN DOWN.

A rating of 2 means that very little normal speech is present.

N.B. A free flow of delusions is not necessarily incoherent. A subject may talk about delusions quite coherently.

Flight of ideas. Words are associated together inappropriately by sound or rhyme (clang association). Although the original aim of the sentence may quickly be lost, a path can be traced through associations of the white–black–coffin or ring–wrong variety, or through associations with distracting stimuli, e.g. ☐ (137)

'How is your appetite?' 'I feel as if I have lost my appetite. I have had an orange. A real juicy orange.' (Sees patient walking past window.) 'She is going for E.C.T. Etcetera treatment or teddy bear's picnic. I call it.'

DO NOT RATE THIS SYMPTOM PRESENT UNLESS EXAMPLES ARE WRITTEN DOWN.

A rating of 2 means that very little normal speech is present.

Poverty of content of speech. The subject talks freely but so vaguely that little information is given in spite of the number of words used: rambles on without coming to a point; may wander far from original theme. Exclude incoherence or flight of ideas. Rate only if severe and always give written example. ☐ (138)

Misleading answers. Subject's answers are misleading because answers 'yes' or 'no' to everything, or frequent self-contradictions, or appears to be deliberately misleading. Do not include incoherence, flight of ideas or poverty of speech here. ☐ (139)

Re-rate adequacy of interview ☐ (140)

0 = Ratings made adequately represent the symptoms present.
1 = Some problem but key symptoms have been rated.
2 = Serious question as to adequacy of interview for rating key symptoms (other than sections 18–20).
3 = *Only* sections 18–20 could be rated.

Check that every box has an entry except those below ticked cut-off points.

Complete coding sheet if one is being used.

APPENDIX V

Beck Depression Inventory

BECK DEPRESSION INVENTORY

NAME: SEX:

AGE: MARITAL STATUS:

Here are some statements regarding the way people feel and think. The statements are grouped in 21 sections from A to U. One statement must be chosen from each section. You are requested to put a circle round the number of the statement which fits you best at the moment.

A 0 I do not feel sad

1 I feel blue or sad

2a I am blue or sad all the time and I can't snap out of it

2b I am so sad or unhappy that it is very painful

3 I am so sad and unhappy that I can't stand it

B 0 I am not particularly pessimistic or discouraged about the future

1a I feel discouraged about the future

2a I feel I have nothing to look forward to

2b I feel that I won't ever get over my troubles

3 I feel that the future is hopeless and that things cannot improve

C 0 I do not feel like a failure

1 I feel that I have failed more than average

2a I feel I have accomplished very little that is worthwhile or that means anything

2b As I look back on my life all I can see is a lot of failures

3 I feel I am a complete failure as a person (parent, husband, wife)

D	0	I am not particularly dissatisfied
	1a	I feel bored most of the time
	1b	I don't enjoy things the way I used to
	2	I don't get satisfaction out of anything any more
	3	I am dissatisfied with everything
E	0	I don't feel particularly guilty
	1	I feel bad or unworthy a good part of the time
	2a	I feel quite guilty
	2b	I feel bad or unworthy practically all the time now
	3	I feel as though I am very bad or worthless
F	0	I don't feel I am being punished
	1	I have a feeling that something bad may happen to me
	2	I feel I am being punished or will be punished
	3a	I feel I deserve to be punished
	3b	I want to be punished
G	0	I don't feel disappointed in myself
	1a	I am disappointed in myself
	1b	I don't like myself
	2	I am disgusted with myself
	3	I hate myself
H	0	I don't feel I am worse than anyone else
	1	I am very critical of myself for my weaknesses, or mistakes
	2a	I blame myself for everything that goes wrong
	2b	I feel I have many bad faults

I	0	I don't have any thoughts of harming myself
	1	I have thoughts of harming myself but I would not carry them out
	2a	I feel I would be better off dead
	2b	I have definite plans about committing suicide
	2c	I feel my family would be better off if I were dead
	3	I would kill myself if I could
J	0	I don't cry any more than usual
	1	I cry more now than I used to
	2	I cry all the time now. I can't stop it
	3	I used to be able to cry but now I can't cry at all even though I want to
K	0	I am no more irritated now than I ever am
	1	I get annoyed or irritated more easily than I used to
	2	I feel irritated all the time
	3	I don't get irritated at all at the things that used to irritate me
L	0	I have not lost interest in other people
	1	I am less interested in other people now than I used to be
	2	I have lost most of my interest in other people and have little feelings for them
	3	I have lost all my interest in other people and don't care about them at all
M	0	I make decisions about as well as ever
	1	I am less sure of myself now and try to put off making decisions
	2	I can't make decisions any more without help
	3	I can't make any decisions at all any more

N 0 I don't feel I look any worse than I used to

1 I am worried that I am looking old or unattractive

2 I feel that there are permanent changes in my appearance and they make me look unattractive

3 I feel that I am ugly or repulsive to look at

O 0 I can work about as well as before

1a It takes an extra effort to get started at doing something

1b I don't work as well as I used to

2 I have to push myself very hard to do anything

3 I can't do any work at all

P 0 I can sleep as well as usual

1 I wake up more tired in the morning than I used to

2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep

3 I wake up early every day and can't get more than 5 hours' sleep

Q 0 I don't get any more tired than usual

1 I get tired more easily than I used to

2 I get tired from doing anything

3 I get too tired to do anything

R 0 My appetite is no worse than usual

1 My appetite is not as good as it used to be

2 My appetite is much worse now

3 I have no appetite at all any more

S 0 I haven't lost much weight, if any, lately

1 I have lost more than 5 lbs

2 I have lost more than 10 lbs

3 I have lost more than 15 lbs

T 0 I am no more concerned about my health than usual

1 I am concerned about aches and pains or upset stomach or constipation or other unpleasant feelings in my body

2 I am so concerned with how I feel or what I feel that it's hard to think of much else

3 I am completely absorbed in what I feel

U 0 I have not noticed any recent change in my interest in sex

1 I am less interested in sex than I used to be

2 I am much less interested in sex now

3 I have lost interest in sex completely

APPENDIX VI

Irritability, depression, anxiety scale

This questionnaire is to help the doctor to know how you are feeling at present. Read each item carefully and underline the response which best shows how you are feeling now, or have been feeling in the last day or two.

A clinical scale for the self-assessment of irritability

1. I feel cheerful

   Yes, definitely
   Yes, sometimes
   No, not much
   No, not at all

2. I can sit down and relax quite easily

   Yes, definitely
   Yes, sometimes
   No, not much
   No, not at all

3. My appetite is

   very poor
   fairly good
   quite good
   very good

4. I lose my temper and shout and snap at others

   Yes, definitely
   Yes, sometimes
   No, not much
   No, not at all

5. I feel tense or wound up

   Yes, definitely
   Yes, sometimes
   No, not much
   No, not at all

6. I feel like harming myself

   Yes, definitely
   Yes, sometimes
   No, not much
   No, not at all

7. I have kept up my old interests

Yes, most of them
Yes, some of them
No, not many of them
No, none of them

8. I am patient with other people

All of the time
Most of the time
Some of the time
Hardly ever

9. I get scared or panicky for no very good reason

Yes, definitely
Yes, sometimes
No, not much
Not at all

10. I get angry with myself or call myself names

Yes, definitely
Sometimes
Not often
No, not at all

11. I can laugh and feel amused

Yes, definitely
Yes, sometimes
No, not much
No, not at all

12. I feel I might lose control and hit or hurt somebody

Sometimes
Occasionally
Rarely
Never

13. I have an uncomfortable feeling like butterflies in my stomach

Yes, definitely
Yes, sometimes
Not very often
Not at all

14. The thought of hurting myself occurs to me

Sometimes
Not very often
Hardly ever
Not at all

15. I'm awake before I need to get up

For two hours or more
For about one hour
For less than one hour
Not at all, I sleep until it is time to get up

16. People upset me so that I feel like slamming doors or banging about

Yes, often
Yes, sometimes
Only occasionally
Not at all

17. I can go out on my own without feeling anxious

Yes, always
Yes, sometimes
No, not often
No, I never can

18. Lately I have been getting annoyed with myself

Very much so
Rather a lot
Not much
Not at all

APPENDIX VII

Semantic differential

Semantic Differential

The purpose of this exercise is to measure the meaning of certain things to various people by having them judge them against a series of descriptive scales. In doing this please make your judgement on the basis of what these mean to you. On each page of this pamphlet you will find a different concept to be judged and beneath it a set of scales. You are to rate the concept on each of these scales in order.

Here is how you are to use these scales:

If you feel that the concept at the top of the page is very closely related to one end of the scale, you should place your check-mark as follows:

fair ___X___:_____:_____:_____:_____:_____:_____: unfair

fair _____:_____:_____:_____:_____:_____:___X___: unfair

If you feel that the concept is quite closely related to one or the other end of the scale (but not extremely), you should place your check-mark as follows:

strong _____:___X___:_____:_____:_____:_____:_____: weak

strong _____:_____:_____:_____:_____:___X___:_____: weak

If the concept seems only slightly related to one side as opposed to the other side (but is not really neutral), then you should check as follows:

active _____:_____:___X___:_____:_____:_____:_____: passive

active _____:_____:_____:_____:___X___:_____:_____: passive

The direction towards which you check, of course, depends upon which of the two ends of the scale seems most characteristic of the things you are judging.

If you consider the concept to be neutral on the scale, both sides of the scale equally associated with the concept, or if the scale is completely irrelevant, unrelated to the concept, then you should place your check-mark in the middle space.

safe _____:_____:_____:___X___:_____:_____:_____: dangerous

Never put more than one check-mark on a single scale.

Sometimes you may feel as though you have had the same item before on the test. This will not be the case, so do not look back and forth through the items. Do not try to remember how you checked similar items earlier in the test. Make each item a separate and independent judgement. Work at a steady pace and don't worry or puzzle much over individual items. It is your first impressions, the immediate 'feelings' about the items that we want. On the other hand, please avoid carelessness, because we want your true impressions.

Me as I am now

1.	successful	____:	____:	____:	____:	____:	____:	____:	unsuccessful
2.	bad	____:	____:	____:	____:	____:	____:	____:	good
3.	worthless	____:	____:	____:	____:	____:	____:	____:	worthwhile
4.	hopeful	____:	____:	____:	____:	____:	____:	____:	hopeless
5.	active	____:	____:	____:	____:	____:	____:	____:	passive
6.	interesting	____:	____:	____:	____:	____:	____:	____:	dull
7.	slow	____:	____:	____:	____:	____:	____:	____:	fast
8.	unpleasant	____:	____:	____:	____:	____:	____:	____:	pleasant
9.	negative	____:	____:	____:	____:	____:	____:	____:	positive
10.	in control	____:	____:	____:	____:	____:	____:	____:	helpless
11.	inadequate	____:	____:	____:	____:	____:	____:	____:	adequate
12.	happy	____:	____:	____:	____:	____:	____:	____:	sad

Me as I was before this illness

1.	successful	____:	____:	____:	____:	____:	____:	____:	unsuccessful
2.	bad	____:	____:	____:	____:	____:	____:	____:	good
3.	worthless	____:	____:	____:	____:	____:	____:	____:	worthwhile
4.	hopeful	____:	____:	____:	____:	____:	____:	____:	hopeless
5.	active	____:	____:	____:	____:	____:	____:	____:	passive
6.	interesting	____:	____:	____:	____:	____:	____:	____:	dull
7.	slow	____:	____:	____:	____:	____:	____:	____:	fast
8.	unpleasant	____:	____:	____:	____:	____:	____:	____:	pleasant
9.	negative	____:	____:	____:	____:	____:	____:	____:	positive
10.	in control	____:	____:	____:	____:	____:	____:	____:	helpless
11.	inadequate	____:	____:	____:	____:	____:	____:	____:	adequate
12.	happy	____:	____:	____:	____:	____:	____:	____:	sad

Me as I would like to be

1.	successful	____:	____:	____:	____:	____:	____:	____:	unsuccessful
2.	bad	____:	____:	____:	____:	____:	____:	____:	good
3.	worthless	____:	____:	____:	____:	____:	____:	____:	worthwhile
4.	hopeful	____:	____:	____:	____:	____:	____:	____:	hopeless
5.	active	____:	____:	____:	____:	____:	____:	____:	passive
6.	interesting	____:	____:	____:	____:	____:	____:	____:	dull
7.	slow	____:	____:	____:	____:	____:	____:	____:	fast
8.	unpleasant	____:	____:	____:	____:	____:	____:	____:	pleasant
9.	negative	____:	____:	____:	____:	____:	____:	____:	positive
10.	in control	____:	____:	____:	____:	____:	____:	____:	helpless
11.	inadequate	____:	____:	____:	____:	____:	____:	____:	adequate
12.	happy	____:	____:	____:	____:	____:	____:	____:	sad

My environment as it is now

1.	successful	____:	____:	____:	____:	____:	____:	____:	unsuccessful
2.	bad	____:	____:	____:	____:	____:	____:	____:	good
3.	worthless	____:	____:	____:	____:	____:	____:	____:	worthwhile
4.	hopeful	____:	____:	____:	____:	____:	____:	____:	hopeless
5.	active	____:	____:	____:	____:	____:	____:	____:	passive
6.	interesting	____:	____:	____:	____:	____:	____:	____:	dull
7.	slow	____:	____:	____:	____:	____:	____:	____:	fast
8.	unpleasant	____:	____:	____:	____:	____:	____:	____:	pleasant
9.	negative	____:	____:	____:	____:	____:	____:	____:	positive
10.	in control	____:	____:	____:	____:	____:	____:	____:	helpless
11.	inadequate	____:	____:	____:	____:	____:	____:	____:	adequate
12.	happy	____:	____:	____:	____:	____:	____:	____:	sad

The future as I see it for me

1.	successful	____:	____:	____:	____:	____:	____:	____:	unsuccessful
2.	bad	____:	____:	____:	____:	____:	____:	____:	good
3.	worthless	____:	____:	____:	____:	____:	____:	____:	worthwhile
4.	hopeful	____:	____:	____:	____:	____:	____:	____:	hopeless
5.	active	____:	____:	____:	____:	____:	____:	____:	passive
6.	interesting	____:	____:	____:	____:	____:	____:	____:	dull
7.	slow	____:	____:	____:	____:	____:	____:	____:	fast
8.	unpleasant	____:	____:	____:	____:	____:	____:	____:	pleasant
9.	negative	____:	____:	____:	____:	____:	____:	____:	positive
10.	in control	____:	____:	____:	____:	____:	____:	____:	helpless
11.	inadequate	____:	____:	____:	____:	____:	____:	____:	adequate
12.	happy	____:	____:	____:	____:	____:	____:	____:	sad

APPENDIX VIII

Hopelessness Scale
(General Expectancy Scale)

Here are some statements about the way you see the future. Read each statement carefully. If the statement describes how you think about the future, circle the word true at the side of the questionnaire. If it does not describe how you think about the future, circle the word false at the side of the questionnaire.

1.	I look forward to the future with hope and enthusiasm	True	False
2.	I might as well give up because I can't make things better for myself	True	False
3.	When things are going badly I am helped by knowing that they can't stay that way for ever	True	False
4.	I can't imagine what my life would be like in 10 years	True	False
5.	I have enough time to accomplish the things I most want to do	True	False
6.	In the future I expect to succeed in what concerns me most	True	False
7.	My future seems dark to me	True	False
8.	I expect to get more of the good things in life than the average person	True	False
9.	I just don't get the breaks, and there's no reason to believe I will in the future	True	False
10.	My past experiences have prepared me well for my future	True	False
11.	All I can see ahead of me is unpleasantness rather than pleasantness	True	False
12.	I don't expect to get what I really want	True	False
13.	When I look ahead to the future I expect that I will be happier than I am now	True	False
14.	Things just don't work out the way I want them to	True	False
15.	I have great faith in the future	True	False
16.	I never get what I want so it is foolish to want anything	True	False
17.	It is very unlikely that I will get any real satisfaction in the future	True	False
18.	The future seems vague and uncertain to me	True	False
19.	I can look forward to more good times than bad times	True	False
20.	There's no use in really trying to get something I want because I probably won't get it	True	False

APPENDIX IX

17 Item Hamilton Rating Scale for Depression

HAMILTON DEPRESSION SCALE

NAME: DATE:

Item no.	Score range	Symptom	Score
1	0–4	Depressed mood	
2	0–4	Guilt	
3	0–4	Suicide	
4	0–2	Insomnia, initial	
5	0–2	Insomnia, middle	
6	0–2	Insomnia, delayed	
7	0–4	Work and interests	
8	0–4	Retardation	
9	0–4	Agitation	
10	0–4	Anxiety, psychic	
11	0–4	Anxiety, somatic	
12	0–2	Somatic symptoms, gastro-intestinal	
13	0–2	Somatic symptoms, general	
14	0–2	Somatic symptoms, genital	
15	0–4	Hypochondriasis	
16	0–2	Loss of insight	
17	0–2	Loss of weight	
18	0–2	Diurnal variation $\frac{M}{E}$	
19	0–4	Depersonalisation, etc.	
20	0–2	Obsessional symptoms	

Grading

0	Absent
1	Mild or trivial
2 3	Moderate
4	Severe

0	Absent
1	Slight or doubtful
2	Clearly present

N.B. Items 18, 19, 20 were rated but not used in the analysis

APPENDIX X

Example of voice print in recording speech rate

APPENDIX XI

List of PSE items used to meet the research diagnostic criteria for primary major depression

Presence of reported symptoms in the hospital and general practice populations

APPENDIX XI

Item	Symptom	Rating	
23	depressed mood	1 or 2	
19	inefficient thinking	1 or 2	either symptom
20	poor concentration	1 or 2	
22	loss of interest	1 or 2	any one symptom
38	loss of libido	1 or 2	
28	avoidance of social contact	1 or 2	
35	delayed sleep	1 or 2	either symptom
37	early wakening	1 or 2	
34	loss of weight	1 or 2	
29	self-depreciation	1 or 2	either symptom
33	self-blame	1 or 2	
25	suicidal plans or acts	1,2 or 3	
110	slowness and underactivity	1 only	
111	agitation	1 only	
6	loss of energy, tired	1 or 2	either symptom
36	subjective anergia	1 or 2	
26	clinical judgement, i.e. primacy of depression	1 or 2	

Present State Examination (PSE)

Presence or absence of reported symptoms in the hospital population (N = 49) and general practice population (N = 38, 1 PSE missing)

PSE item	symptom			Hospital Frequency	Hospital %	G.P. Frequency	G.P. %
1	physical health	rating:	(1)	22	45	18	47
			(2)	9	18	6	16
			(3)	3	.07	0	0
2	doctor's opinion	rating:	(1)	10	21	2	.05
			(2)	2	4	0	0
			(3)	0	0	1	.02
3	psychosomatic			1	.02	1	.02
4	worrying			42	86	30	79
5	tension pains			25	51	23	61
6	loss of energy, exhaustion			43	88	34	89
7	muscular tension			33	67	22	58
8	agitation			24	49	18	47
9	hypochondriasis			8	16	6	16
10	psychic tension			44	90	33	87
11	autonomic tension			34	69	24	63
12	anxious foreboding			8	16	11	29
13	anxiety due to delusions			2	.04	0	0
14	panic attacks			10	20	1	.02
15	situational anxiety			15	31	3	.08
16	anxiety over meeting people			20	41	4	11
17	specific phobias			7	14	2	.05
18	anxiety avoidance			5	10	4	11
19	inefficient thinking			37	76	23	61
20	poor concentration			36	73	28	74
21	neglect because of brooding			28	57	22	58
22	loss of interest in activities			40	82	24	63
23	depressed mood			49	100	38	100
24	hopelessness			43	88	33	87
25	suicidal ideas or acts			27	55	17	45
26	primary depression			38	78	30	79
	anxiety and depression			12	24	7	18
27 (rated 2)	diurnal variation			22	45	12	32
28	avoidance of social contact			36	73	24	63
29	self-depreciation			34	69	20	53
30	lack of self confidence			41	84	24	63
31	simple ideas of reference			15	31	10	26
32	guilty ideas of reference			8	16	8	21
33	self-blame			28	57	18	47
34	weight loss			18	37	12	32
35	delayed sleep			22	45	22	58
36	subjective anergia			42	86	24	63

PSE item	symptom	Hospital		G.P.	
		Frequency	%	Frequency	%
37	early morning wakening	19	39	21	55
38	loss of libido	23	47	9	24
39	premenstrual exacerbation	12	24	10	26
40	irritability	43	87	33	87
41	expansive mood	2	.04	0	0
42	ideomotor pressure	1	.02	0	0
43	grandiose ideas	0	0	0	0
44	obsessional checking	10	20	4	11
45	obsessional cleanliness	4	.08	0	0
46	obsessional ideas	13	27	4	11
47	derealisation	5	10	0	0
48	depersonalisation	8	16	0	0
100	dissociative state	3	.06	0	0
101	conversion symptom	1	.02	0	0
110	slowness, underactivity	4	.08	2	.05
111	agitation	5	10	1	.02
120	observed anxiety	15	31	14	37
121	observed depression	42	86	27	71
122	hystrionic	3	.06	1	.02
130	slowed speech	5	10	0	0

APPENDIX XII

Two-way analysis of variance for the entire population (hospital and general practice)

APPENDIX XII

AGE

Source of variation	F ratio	Df
LOC	3.262	1
TR	1.650	2
LOC x TR	1.065	2
Explained	1.718	5
Residual		82
Total		87

EDUC

Source of variation	F ratio	Df
LOC	20.698	1
TR	0.498	2
LOC x TR	1.397	2
Explained	4.890	5
Residual		82
Total		87

SOC

Source of variation	F ratio	Df
LOC	24.955	1
TR	0.700	2
LOC x TR	0.408	2
Explained	5.408	5
Residual		82
Total		87

DILL

Source of variation	F ratio	Df
LOC	11.131	1
TR	3.478	2
LOC x TR	4.039	2
Explained	5.174	5
Residual		82
Total		87

PSE

Source of variation	F ratio	Df
LOC	10.277	1
TR	0.302	2
LOC x TR	0.703	2
Explained	2.475	5
Residual		78
Total		83

BDI

Source of variation	F ratio	Df
LOC	0.285	1
TR	0.830	2
LOC x TR	2.295	2
Explained	1.316	5
Residual		78
Total		85

HS

Source of variation	F ratio	Df
LOC	3.165	1
TR	0.079	2
LOC x TR	0.476	2
Explained	0.855	5
Residual		78
Total		83

DEP

Source of variation	F ratio	Df
LOC	0.810	1
TR	0.263	2
LOC x TR	0.715	2
Explained	0.548	5
Residual		78
Total		83

ANX

Source of variation	F ratio	Df
LOC	0.300	1
TR	0.102	2
LOC x TR	0.260	2
Explained	0.208	5
Residual		78
Total		83

IN

Source of variation	F ratio	Df
LOC	0.177	1
TR	0.079	2
LOC x TR	0.143	2
Explained	0.124	5
Residual		79
Total		84

OUT

Source of variation	F ratio	Df
LOC	3.450	1
TR	1.252	2
LOC x TR	3.015	2
Explained	2.373	5
Residual		79
Total		84

IRR

Source of variation	F ratio	Df
LOC	0.769	1
TR	0.616	2
LOC x TR	1.605	2
Explained	1.035	5
Residual		79
Total		84

AM

Source of variation	F ratio	Df
LOC	5.280	1
TR	0.664	2
LOC x TR	0.585	2
Explained	1.569	5
Residual		79
Total		84

WAS

Source of variation	F ratio	Df
LOC	0.176	1
TR	0.581	2
LOC x TR	1.535	2
Explained	0.884	5
Residual		79
Total		84

WD

Source of variation	F ratio	Df
LOC	0.471	1
TR	1.280	2
LOC x TR	2.158	2
Explained	1.476	5
Residual		79
Total		84

ENV

Source of variation	F ratio	Df
LOC	0.181	1
TR	0.865	2
LOC x TR	0.673	2
Explained	0.650	5
Residual		79
Total		84

FUT

Source of variation	F ratio	Df
LOC	1.019	1
TR	0.691	2
LOC x TR	1.589	2
Explained	1.109	5
Residual		79
Total		84

IDS

Source of variation	F ratio	Df
LOC	2.759	1
TR	1.411	2
LOC x TR	2.212	2
Explained	2.004	5
Residual		79
Total		84

IDW

Source of variation	F ratio	Df
LOC	0.035	1
TR	0.256	2
LOC x TR	0.343	2
Explained	0.248	5
Residual		79
Total		84

WASE

Source of variation	F ratio	Df
LOC	1.485	1
TR	1.232	2
LOC x TR	2.424	2
Explained	1.783	5
Residual		72
Total		77

HRS

Source of variation	F ratio	Df
LOC	1.998	1
TR	4.162	2
LOC x TR	0.181	2
Explained	2.095	5
Residual		72
Total		77

CS

Source of variation	F ratio	Df
LOC	1.071	1
TR	0.065	2
LOC x TR	0.427	2
Explained	0.410	5
Residual		72
Total		77

WS

Source of variation	F ratio	Df
LOC	0.307	1
TR	3.487	2
LOC x TR	1.098	2
Explained	1.871	5
Residual		72
Total		77

APPENDIX XIII

TWO-WAY ANALYSIS OF VARIANCE FOR THE COMPLETERS AND NONCOMPLETERS

APPENDIX XIII

AGE

Source of variation	F ratio	Df
STAT	3.867	1
TR	1.621	2
STAT x TR	0.411	2
Explained	1.573	5
Residual		82
Total		87

EDUC

Source of variation	F ratio	Df
STAT	3.986	1
TR	0.375	2
STAT x TR	1.662	2
Explained	1.624	5
Residual		82
Total		87

SOC

Source of variation	F ratio	Df
STAT	1.398	1
TR	0.466	2
STAT x TR	1.190	2
Explained	0.951	5
Residual		82
Total		87

DILL

Source of variation	F ratio	Df
STAT	0.045	1
TR	2.700	2
STAT x TR	0.172	2
Explained	1.162	5
Residual		82
Total		87

PSE

Source of variation	F ratio	Df
STAT	0.281	1
TR	0.291	2
STAT x TR	0.651	2
Explained	0.439	5
Residual		78
Total		83

BDI

Source of variation	F ratio	Df
STAT	25.955	1
TR	1.378	2
STAT x TR	1.638	2
Explained	6.288	5
Residual		78
Total		83

HS

Source of variation	F ratio	Df
STAT	2.527	1
TR	0.045	2
STAT x TR	0.100	2
Explained	0.576	5
Residual		78
Total		83

DEP

Source of variation	F ratio	Df
STAT	3.344	1
TR	0.347	2
STAT x TR	2.154	2
Explained	1.637	5
Residual		78
Total		83

ANX

Source of variation	F ratio	Df
STAT	2.203	1
TR	0.149	2
STAT x TR	0.365	2
Explained	0.632	5
Residual		78
Total		83

IN

Source of variation	F ratio	Df
STAT	5.178	1
TR	0.043	2
STAT x TR	0.719	2
Explained	1.357	5
Residual		79
Total		84

OUT

Source of variation	F ratio	Df
STAT	2.153	1
TR	1.043	2
STAT x TR	0.743	2
Explained	1.174	5
Residual		79
Total		84

IRR

Source of variation	F ratio	Df
STAT	5.073	1
TR	0.527	2
STAT x TR	1.083	2
Explained	1.697	5
Residual		79
Total		84

AM

Source of variation	F ratio	Df
STAT	3.371	1
TR	0.757	2
STAT x TR	0.065	2
Explained	0.970	5
Residual		79
Total		84

WAS

Source of variation	F ratio	Df
STAT	0.044	1
TR	0.555	2
STAT x TR	0.083	2
Explained	0.269	5
Residual		79
Total		84

WD

Source of variation	F ratio	Df
STAT	0.066	1
TR	1.231	2
STAT x TR	0.995	2
Explained	0.913	5
Residual		79
Total		84

ENV

Source of variation	F ratio	Df
STAT	6.020	1
TR	1.085	2
STAT x TR	0.172	2
Explained	1.638	5
Residual		79
Total		84

FUT

Source of variation	F ratio	Df
STAT	2.037	1
TR	0.748	2
STAT x TR	0.778	
Explained	0.986	5
Residual		79
Total		84

IDS

Source of variation	F ratio	Df
STAT	2.472	1
TR	1.492	2
STAT x TR	0.587	2
Explained	1.273	5
Residual		79
Total		84

IDW

Source of variation	F ratio	Df
STAT	0.165	1
TR	0.254	2
STAT x TR	0.041	2
Explained	0.153	5
Residual		79
Total		84

WASE

Source of variation	F ratio	Df
STAT	1.476	1
TR	1.394	2
STAT x TR	0.025	2
Explained	0.790	5
Residual		72
Total		77

HRS

Source of variation	F ratio	Df
STAT	8.575	1
TR	5.359	2
STAT x TR	1.056	2
Explained	3.943	5
Residual		72
Total		77

CS

Source of variation	F ratio	Df
STAT	0.424	1
TR	0.081	2
STAT x TR	0.785	2
Explained	0.424	5
Residual		72
Total		77

WS

Source of variation	F ratio	Df
STAT	0.047	1
TR	3.455	2
STAT x TR	1.719	2
Explained	2.090	5
Residual		72
Total		77

APPENDIX XIV

Two-way analysis of variance
for completers only

(hospital and general practice)

APPENDIX XIV

AGE

Source of variation	F ratio	Df
LOC	1.554	1
TR	2.050	2
LOC x TR	1.411	2
Explained	1.674	5
Residual		58
Total		63

EDUC

Source of variation	F ratio	Df
LOC	23.137	1
TR	0.834	2
LOC x TR	0.742	2
Explained	5.333	5
Residual		58
Total		63

SOC

Source of variation	F ratio	Df
LOC	24.103	1
TR	0.696	2
LOC x TR	0.239	2
Explained	5.223	5
Residual		58
Total		63

DILL

Source of variation	F ratio	Df
LOC	6.406	1
TR	2.686	2
LOC x TR	1.803	2
Explained	3.022	5
Residual		58
Total		63

PSE

Source of variation	F ratio	Df
LOC	4.102	1
TR	0.077	2
LOC x TR	0.681	2
Explained	1.131	5
Residual		57
Total		62

BDI

Source of variation	F ratio	Df
LOC	0.913	1
TR	1.947	2
LOC x TR	1.593	2
Explained	1.587	5
Residual		57
Total		62

HS

Source of variation	F ratio	Df
LOC	4.040	1
TR	0.058	2
LOC x TR	0.103	2
Explained	0.888	5
Residual		57
Total		62

DEP

Source of variation	F ratio	Df
LOC	0.576	1
TR	1.392	2
LOC x TR	0.612	2
Explained	0.925	5
Residual		57
Total		62

ANX

Source of variation	F ratio	Df
LOC	0.038	1
TR	0.326	2
LOC x TR	0.179	2
Explained	0.208	5
Residual		57
Total		62

IN

Source of variation	F ratio	Df
LOC	0.006	1
TR	0.047	2
LOC x TR	0.068	2
Explained	0.047	5
Residual		58
Total		63

OUT

Source of variation	F ratio	Df
LOC	7.423	1
TR	0.769	2
LOC. x TR	1.298	2
Explained	2.275	5
Residual		58
Total		63

IRR

Source of variation	F ratio	Df
LOC	2.463	1
TR	0.259	2
LOC x TR	0.703	2
Explained	0.871	5
Residual		58
Total		63

AM

Source of variation	F ratio	Df
LOC	4.146	1
TR	0.909	2
LOC x TR	0.852	2
Explained	1.506	5
Residual		58
Total		63

WAS

Source of variation	F ratio	Df
LOC	0.530	1
TR	0.641	2
LOC x TR	1.016	2
Explained	0.786	5
Residual		58
Total		63

WD

Source of variation	F ratio	Df
LOC	0.012	1
TR	2.466	2
LOC x TR	1.073	2
Explained	1.425	5
Residual		58
Total		63

ENV

Source of variation	F ratio	Df
LOC	0.729	1
TR	1.266	2
LOC x TR	1.615	2
Explained	1.321	5
Residual		58
Total		63

FUT

Source of variation	F ratio	Df
LOC	2.862	1
TR	1.339	2
LOC x TR	0.523	2
Explained	1.264	5
Residual		58
Total		63

IDS

Source of variation	F ratio	Df
LOC	3.013	1
TR	2.215	2
LOC x TR	1.124	2
Explained	1.889	5
Residual		58
Total		63

IDW

Source of variation	F ratio	Df
LOC	0.581	1
TR	0.264	2
LOC x TR	0.482	2
Explained	0.420	5
Residual		58
Total		63

WASE

Source of variation	F ratio	Df
LOC	1.059	1
TR	1.397	2
LOC x TR	1.959	2
Explained	1.535	5
Residual		56
Total		61

HRS

Source of variation	F ratio	Df
LOC	1.815	1
TR	6.179	2
LOC x TR	0.378	2
Explained	3.042	5
Residual		56
Total		61

CS

Source of variation	F ratio	Df
LOC	0.294	1
TR	0.053	2
LOC x TR	0.204	2
Explained	0.162	5
Residual		56
Total		61

WS

Source of variation	F ratio	Df
LOC	0.141	1
TR	2.560	2
LOC x TR	0.166	2
Explained	1.118	5
Residual		56
Total		61

APPENDIX XV

List of symptoms used to meet the research diagnostic criteria for endogenous major depression

APPENDIX XV

Research Diagnostic Criteria: Endogenous Major Depressive Disorder

A. 1. Distinct quality of depressed mood

2. Lack of reactivity to environmental changes

3. Mood is worse a.m.

4. Pervasive loss of interest or pleasure.

B. 1. Feelings of self-reproach or excessive or inappropriate guilt

2. Early morning awakening or middle insomnia

3. Psychomotor retardation or agitation (more than mere subjective feeling)

4. Poor appetite

5. Weight loss (2 lbs a week over several weeks or 20 lbs a year, not on a diet)

6. Loss of interest or pleasure (may or may not be pervasive) in usual activities or decreased sexual drive.

(From groups A and B a total of at least four symptoms for probable, 6 for definite; including at least one from group A)

(Ignore precipitating events)

APPENDIX XVI

Two-way analysis of covariance for change scores

APPENDIX XV I

Change in BDI

Source of variation	F ratio	Df
Covariates	2.484	4
EDUC	0.737	1
SOC	0.355	1
DILL	8.155	1
PSE	1.960	1
LOC	2.140	1
TR	4.239	2
LOC x TR	4.476	2
Explained	3.202	9
Residual		53
Total		62

Change in HS

Source of variation	F ratio	Df
Covariates	1.194	4
EDUC	1.069	1
SOC	1.534	1
DILL	1.618	1
PSE	2.186	1
LOC	0.576	1
TR	1.681	2
LOC x TR	0.098	2
Explained	0.957	9
Residual		53
Total		62

Change in DEP

Source of variation	F ratio	Df
Covariates	3.538	4
EDUC	1.832	1
SOC	0.071	1
DILL	13.399	1
PSE	0.030	1
LOC	0.214	1
TR	2.232	2
LOC x TR	0.576	2
Explained	2.203	9
Residual		53
Total		62

Change in ANX

Source of variation	F ratio	Df
Covariates	1.683	4
EDUC	0.493	1
SOC	0.022	1
DILL	6.575	1
PSE	0.033	1
LOC	2.620	1
TR	6.093	2
LOC x TR	0.087	2
Explained	2.295	9
Residual		53
Total		62

Change in IN

Source of variation	F ratio	Df
Covariates	1.813	4
EDUC	0.365	1
SOC	0.965	1
DILL	4.509	1
PSE	1.415	1
LOC	0.295	1
TR	4.577	2
LOC x TR	0.513	2
Explained	1.941	9
Residual		53
Total		62

Change in OUT

Source of variation	F ratio	Df
Covariates	1.529	4
EDUC	0.180	1
SOC	1.505	1
DILL	0.240	1
PSE	1.384	1
LOC	0.689	1
TR	0.518	2
LOC x TR	0.423	2
Explained	0.987	9
Residual		54
Total		63

Change in IRR

Source ov variation	F ratio	Df
Covariates	1.682	4
EDUC	0.476	1
SOC	0.001	1
DILL	2.928	1
PSE	2.014	1
LOC	0.011	1
TR	3.339	2
LOC x TR	0.608	2
Explained	1.641	9
Residual		54
Total		63

Change in AM

Source of variation	F ratio	Df
Covariates	4.596	4
EDUC	1.755	1
SOC	2.380	1
DILL	6.869	1
PSE	10.978	1
LOC	0.720	1
TR	4.929	2
LOC x TR	2.430	2
Explained	3.714	9
Residual		54
Total		63

Change in WAS

Source of variation	F ratio	Df
Covariates	0.363	4
EDUC	0.572	1
SOC	0.250	1
DILL	0.993	1
PSE	0.226	1
LOC	0.396	1
TR	0.001	2
LOC x TR	0.439	2
Explained	0.303	9
Residual		54
Total		63

Change in WD

Source of variation	F ratio	Df
Covariates	0.617	4
EDUC	0.539	1
SOC	0.000	1
DILL	0.259	1
PSE	1.696	1
LOC	0.530	1
TR	0.333	2
LOC x TR	1.984	2
Explained	0.837	9
Residual		54
Total		63

Change in ENV

Source of variation	F ratio	Df
Covariates	3.627	4
EDUC	1.353	1
SOC	8.527	1
DILL	2.866	1
PSE	1.255	1
LOC	0.017	1
TR	3.630	2
LOC x TR	0.489	2
Explained	2.530	9
Residual		54
Total		63

Change in FUT

Source of variation	F ratio	Df
Covariates	0.684	4
EDUC	0.454	1
SOC	0.344	1
DILL	0.856	1
PSE	1.736	1
LOC	0.889	1
TR	3.123	2
LOC x TR	0.246	2
Explained	1.120	9
Residual		54
Total		63

Change in IDS

Source of variation	F ratio	Df
Covariates	4.208	4
EDUC	2.718	1
SOC	1.989	1
DILL	8.685	1
PSE	2.369	1
LOC	0.065	1
TR	1.182	2
LOC x TR	1.841	2
Explained	2.550	9
Residual		54
Total		63

Change in IDW

Source of variation	F ratio	Df
Covariates	0.410	4
EDUC	0.202	1
SOC	0.224	1
DILL	0.578	1
PSE	1.001	1
LOC	0.121	1
TR	0.050	2
LOC x TR	0.938	2
Explained	0.418	9
Residual		54
Total		63

Change in WASE

Source of variation	F ratio	Df
Covariates	2.722	4
EDUC	0.887	1
SOC	1.307	1
DILL	3.075	1
PSE	7.572	1
LOC	0.013	1
TR	1.364	2
LOC x TR	0.123	2
Explained	1.547	9
Residual		54
Total		63

Change in HRS

Source of variation	F ratio	Df
Covariates	2.845	4
EDUC	0.209	1
SOC	0.263	1
DILL	8.684	1
PSE	1.209	1
LOC	0.777	1
TR	9.245	2
LOC x TR	2.035	2
Explained	3.848	9
Residual		50
Total		59

Change in CS

Source of variation	F ratio	Df
Covariates	1.599	4
EDUC	0.399	1
SOC	0.162	1
DILL	0.603	1
PSE	3.801	1
LOC	2.234	1
TR	1.849	2
LOC x TR	0.355	2
Explained	1.481	9
Residual		50
Total		59

Change in WS

Source of variation	F ratio	Df
Covariates	1.142	4
EDUC	0.000	1
SOC	0.714	1
DILL	0.484	1
PSE	3.130	1
LOC	0.932	1
TR	0.468	2
LOC x TR	0.391	2
Explained	0.796	9
Residual		50
Total		59

APPENDIX XVII

Two-way analysis of covariance for percentage change scores

APPENDIX XVII

Percentage change in BDI

Source of variation	F ratio	Df
Covariates	2.660	4
EDUC	0.762	1
SOC	0.439	1
DILL	9.821	1
PSE	0.000	1
LOC	1.269	1
TR	3.500	2
LOC x TR	4.683	2
Explained	3.085	9
Residual		53
Total		62

Percentage change in HS

Source of variation	F ratio	Df
Covariates	1.023	4
EDUC	0.032	1
SOC	1.822	1
DILL	0.553	1
PSE	0.034	1
LOC	0.020	1
TR	1.460	2
LOC x TR	1.072	2
Explained	1.018	9
Residual		53
Total		62

Percentage change in DEP

Source of variation	F ratio	Df
Covariates	4.320	4
EDUC	2.911	1
SOC	0.113	1
DILL	15.519	1
PSE	0.009	1
LOC	0.015	1
TR	1.189	2
LOC x TR	0.710	2
Explained	2.350	9
Residual		53
Total		62

Percentage change in ANX

Source of variation	F ratio	Df
Covariates	1.907	4
EDUC	0.075	1
SOC	0.692	1
DILL	4.594	1
PSE	1.754	1
LOC	0.411	1
TR	4.737	2
LOC x TR	0.200	2
Explained	1.954	9
Residual		53
Total		62

Percentage change in IN

Source of variation	F ratio	Df
Covariates	2.063	4
EDUC	1.388	1
SOC	0.471	1
DILL	4.349	1
PSE	0.033	1
LOC	0.013	1
TR	0.790	2
LOC x TR	0.148	2
Explained	1.126	9
Residual		53
Total		62

Percentage change in OUT

Source of variation	F ratio	Df
Covariates	1.131	4
EDUC	0.009	1
SOC	1.649	1
DILL	0.018	1
PSE	2.394	1
LOC	0.569	1
TR	1.628	2
LOC x TR	0.764	2
Explained	1.123	9
Residual		54
Total		63

Percentage change in IRR

Source of variation	F ratio	Df
Covariates	1.465	4
EDUC	0.101	1
SOC	0.016	1
DILL	4.063	1
PSE	0.181	1
LOC	1.019	3
TR	2.024	2
LOC x TR	0.445	2
Explained	1.214	9
Residual		54
Total		63

Percentage change in AM

Source of variation	F ratio	Df
Covariates	3.355	4
EDUC	1.800	1
SOC	0.721	1
DILL	3.033	1
PSE	9.735	1
LOC	2.003	1
TR	3.355	2
LOC x TR	1.461	2
Explained	2.744	9
Residual		54
Total		63

Percentage change in WAS

Source of variation	F ratio	Df
Covariates	0.200	4
EDUC	0.224	1
SOC	0.246	1
DILL	0.365	1
PSE	0.272	1
LOC	0.226	1
TR	0.007	2
LOC x TR	0.654	2
Explained	0.263	9
Residual		54
Total		63

Percentage change in WD

Source of variation	F ratio	Df
Covariates	0.676	4
EDUC	0.518	1
SOC	0.001	1
DILL	0.345	1
PSE	1.870	1
LOC	0.627	1
TR	0.153	2
LOC x TR	2.080	2
Explained	0.859	9
Residual		54
Total		63

Percentage change in ENV

Source of variation	F ratio	Df
Covariates	1.433	4
EDUC	0.119	1
SOC	3.347	1
DILL	0.222	1
PSE	0.676	1
LOC	0.525	1
TR	2.627	2
LOC x TR	1.387	2
Explained	1.566	9
Residual		54
Total		63

Percentage change in FUT

Source of variation	F ratio	Df
Covariates	0.685	4
EDUC	0.271	1
SOC	0.045	1
DILL	0.063	1
PSE	2.455	1
LOC	2.311	1
TR	1.923	2
LOC x TR	1.557	2
Explained	1.305	9
Residual		54
Total		63

Percentage change in IDS

Source of variation	F ratio	Df
Covariates	4.580	4
EDUC	2.550	1
SOC	1.395	1
DILL	10.509	1
PSE	1.400	1
LOC	0.287	1
TR	0.760	2
LOC x TR	2.303	2
Explained	2.752	9
Residual		54
Total		63

Percentage change in IDW

Source of variation	F ratio	Df
Covariates	0.715	4
EDUC	1.868	1
SOC	1.018	1
DILL	1.190	1
PSE	0.403	1
LOC	0.025	1
TR	1.203	2
LOC x TR	2.951	2
Explained	1.250	9
Residual		54
Total		63

Percentage change in WASE

Source of variation	F ratio	Df
Covariates	0.737	4
EDUC	0.543	1
SOC	0.857	1
DILL	1.455	
PSE	0.917	1
LOC	0.107	1
TR	0.436	2
LOC x TR	0.839	2
Explained	0.631	9
Residual		54
Total		63

Percentage change in HRS

Source of variation	F ratio	Df
Covariates	1.802	4
EDUC	1.552	1
SOC	0.356	1
DILL	6.478	1
PSE	0.168	1
LOC	0.928	1
TR	5.301	2
LOC x TR	3.024	2
Explained	2.739	9
Residual		50
Total		59

Percentage change in CS

Source of variation	F ratio	Df
Covariates	1.684	4
EDUC	3.670	1
SOC	0.669	1
DILL	0.401	1
PSE	2.197	1
LOC	4.943	1
TR	1.802	2
LOC x TR	0.399	2
Explained	1.833	9
Residual		50
Total		59

Percentage change in WS

Source of variation	F ratio	Df
Covariates	0.564	4
EDUC	0.046	1
SOC	0.085	1
DILL	0.154	1
PSE	2.002	1
LOC	0.667	1
TR	1.372	2
LOC x TR	1.205	2
Explained	0.905	9
Residual		50
Total		59

APPENDIX XVIII

Presence of endogenous symptoms in each population (x^2)

APPENDIX XVIII

Presence of endogenous and non-endogenous symptom pattern in the hospital and general practice samples (completers only)

	Endogenous	Non-endogenous	Total
Hospital	16	24	40
G.P.	11	13	24
Total	27	37	64

$X^2 = .51$ Df = 1 NS

APPENDIX XIX

Extension of multiple regression; discriminant function analyses

APPENDIX XIX

Discriminant Function Analysis

Having identified several variables which appear to predict best response in each treatment group, it was considered desirable to use this information to create three 'predicted treatment groups' (combination, CBT and drugs) and assign patients to these groups on the basis of individual characteristics. The reason for doing this was to find out which treatments, in theory, were best suited to individual patients. Moreover, it was felt that, if the different predicted groups were, in fact, composed of different types of people, then it should be feasible to discriminate these categories statistically using variables other than those which defined the categories. By delineating the boundaries between the predicted treatment groups, it might be possible to learn more about which measurements are most effective in distinguishing between the predicted groups, how best to combine the measurements, and how successfully the distinction can be made. The statistical method used to examine these questions was discriminant function analysis.

Essentially, a discriminant function is a regression equation with a dependent variable that represents group membership. The function maximally discriminates the members of the group and indicates to which group each member probably belongs (Kerlinguer, 1973). In brief, if there are two or more independent variables and members of, for example, two groups, the discriminant function gives the 'best' prediction of the 'correct' group membership of each member of the sample. Thus, the discriminant funtion can be used to assign individuals to groups according to their scores on a number of measures (Kerlinguer, ibid).

For a detailed discussion of discriminant function analysis, the reader is referred to Kerlinguer (1973), Tatsuoka (1970, 1971), Rao (1978) and the Manual for the Statistical Package for the Social Sciences (2nd edition, 1975).

In the present study, three regression equations were produced from the data from each treatment group (p.) which gave a predicted response to treatment (i.e. % change in BDI) for each subject who received that treatment. Next, each of the regression equations was applied to each subject in the other categories (e.g. the equation for combination treatment was applied to each subject in CBT and the equation for CBT to each subject in the drugs group). By examining individual cases in this way it was possible to determine, at least hypothetically, which was predicted to be the best treatment in terms of % change on the BDI. For example, subject A who really got combination therapy might have achieved a 50% reduction in BDI score, only a 20% change had he received drugs, or a 40% change if he had been assigned to CBT alone. There was a predicted score also for the treatment he actually received (i.e. combination therapy) which may or may not have coincided with his actual score.

In this way three predicted groups were created, i.e. one for each treatment, and a discriminant function analysis computed (SPSS, 2nd edition, 1975) in order to (1) distinguish statistically between these categories using variables other than those included in the regression equations and (2) on the basis of weights given to each of the variables which comprised the key function, identify the variables (psychological characteristics) which contributed most towards discriminating the predicted treatment groups.

Of the twenty-five variables included initially in the research, eighteen were used in the discriminant analysis. Two variables, total irritability (IRR) and discrepancy between ideal and premorbid self (IDW) had been analysed, in fact, but had to be dropped because they caused signularity in the within groups covariance matrices. Duration of illness (DILL), educational level (EDUC), view of the environment (ENV), referral source (LOC), and ideal self discrepancy(IDS) were excluded because they were variables which had been identified previously as predictors of response to therapy. Since these variables were used to create the categories being discriminated, it would have been artificial to include them in the computation of the discriminant functions.

As suggested in the discussion (p.), it is recognised that this analysis is essentially a statistical manoeuvre in that the information gained from it applies to abstract categories (predicted treatment groups) rather than actual treatment groups. Nevertheless, it was hoped that data from the discriminant analysis might be relevant to the clinician interested in different ways of conceptualising the selection of patients for specific treatments and raise questions for future research.

Table XIX$_1$ Distribution of cases in the predicted groups based on the regression equations for combination, CBT and drugs

Predicted group	Frequency	Percent
COMB (1)	27	42.2
CBT (2)	26	40.6
DRUGS (3)	11	17.2
TOTAL	64	100

From table XIX_1, of the 64 completers in the trial, 27 cases were assigned to the predicted group for combination treatment, 26 cases to the predicted group for CBT and only 11 cases to the predicted group of pharmacotherapy. In other words, when the predictors for each treatment were used as the standard for selection, over 80% of the patients admitted to the trial were assigned to CBT with or without drugs as preferred treatment.

Table XIX_2 Number and relative importance of functions derived in the discriminant analysis of the three predicted treatment groups

Discriminant	Eigenvalue	Relative %	Cononical correlation	Functions derived	Wilk's Lambda	X^2 Df's	Significance
1	1.36	77.6	0.76	0	0.303	59.0 36	0.009
2	0.39	22.4	0.53	1	0.727	16.4, 17	0.49

Table XIX_2 shows the number and relative importance of functions derived in the discriminant analysis of the three predicted groups (categories). Discriminant function 1, with its relatively high eigenvalue and associated correlation, had considerable discriminative power in terms of its capacity to separate the groups, whereas the second function was relatively weak. Looking at the column 'Lambda' (and its associated X^2 tests of significance), before any functions were removed Lambda was 0.303 which further indicated the discriminating power of function 1. However, after some of the information had been removed by the second function, Lambda increased to the point where X^2 was no longer statistically significant (Lambda = 0.727). Function 2 did not add significantly to discriminating the three groups and can, therefore, be discounted.

From tables XIX_3 and XIX_4, the discriminant analysis for the eighteen variables separated the predicted groups (also labelled 'actual categories') for combination, CBT and drugs at a highly significant level. 80.3% of known cases were correctly classified. All of the members of the 'actual' drug group were correctly discriminated. The most incorrectly classified group was the 'actual' combined treatment and cases from this group were assigned predominently to the CBT discriminant predicted group (28%), though a small proportion were also placed in the discriminant predicted group for drugs (8%). Some CBT cases were, in fact, misclassified but all of these were assigned to the discriminant predicted group for combined treatment.

Table XIX_3 Standardised discriminant coefficients for the predicted groups for the three treatments

Variables		Function 1	Function 2
1.	Sex	-0.178	0.120
2.	Age	-0.025	0.022
3.	SOC	-0.290	-0.079
4.	PSE	0.543	0.130
5.	BDI	-0.308	0.122
6.	HS	0.079	-0.391
7.	DEP	0.000	0.362
8.	ANX	-0.030	-0.482
9.	IN	-0.238	0.621
10.	OUT	-0.326	-0.181
11.	AM	-0.332	-2.700
12.	WAS	0.380	2.270
13.	WD	0.142	-0.199
14.	FUT	0.246	0.231
15.	WASE	-0.684	-2.957
16.	HRS	-0.252	-0.255
17.	CS	-0.135	0.011
18.	WS	0.131	0.400

Looking at function 1 (table XIX_3), the greatest negative discriminants were: perceived negative change in self with illness (WASE), level of self-esteem (AM), outward directed irritability (OUT), self-reported depression (BDI), social class (SOC), and observer rated depression (HRS). Variables showing the greatest positive values were: total severity score on the PSE, pre-morbid self-esteem (WAS) and personal future (FUT).

Table XIX_4 Predicted results based on the discriminant functions for variables 1 to 18. Group 1 refers to Combination treatment, group 2 to CBT, and group 3 to Drugs

Actual categories		No. of	Predicted group membership		
Name	Code	Cases	Group 1	Group 2	Group 3
Comb	Group 1	25	16 (64%)	7 (28%)	2 (8%)
CBT	Group 2	25	3 (12%)	22 (88%)	0 (0%)
D	Group 3	11	0 (0%)	0 (0%)	11 (100%)

N.B. Three cases could not be classified in the analysis

80.3% of known cases correctly classified

Chi square = 59.0, p = < .01

Table XIX_5 Mean discriminant scores for each group on the respective functions

Groups	Function 1	Function 2
COMB (1)	0.309	0.594
CBT (2)	-0.826	-0.256
Drug (3)	1.173	-0.766

Table XIX_5 shows the mean discriminant scores for each group on the respective functions. These data and the distribution of cases around each group centroid (mean) are presented graphically in figure XIX_5.

The results for function 1 indicate that the CBT predicted group, with its negative score, has been most easily separated from the predicted group for drugs which has a relatively high, positive discriminant score. However, the boundary between the combination treatment group and the other two treatments is less distinct and the overlap, particularly between the combined therapy and CBT, is considerable.

Thus, relating the discriminant variables to the appropriate predicted treatment groups may be done as follows: total severity score on the PSE, level of pre-morbid functioning (WAS) and outlook about personal future (FUT) identified the predicted drug therapy group. For the CBT predicted group, perceived deviation from normal (WASE), current self-esteem (AM), outward directed irritability (OUT), self-reported (BDI) and observer-rated depression (HRS), and social class (SOC) were the most decisive discriminant variables.

Because of the overlap between the groups, it is difficult to specify the best discriminants for the predicted combined group. However, as the two pharmacotherapy treatments have positive centroid loadings and are, therefore, more closely represented in reduced space (fig.XIX_5), it is likely that the combined group is better identified by the positively weighted discriminant variables, rather than the negative ones. Thus, both pharmacotherapy groups are most accurately discriminated by ratings on psychiatric symptoms (PSE), pre-morbid functioning (WAS) and appraisal of personal future (FUT).

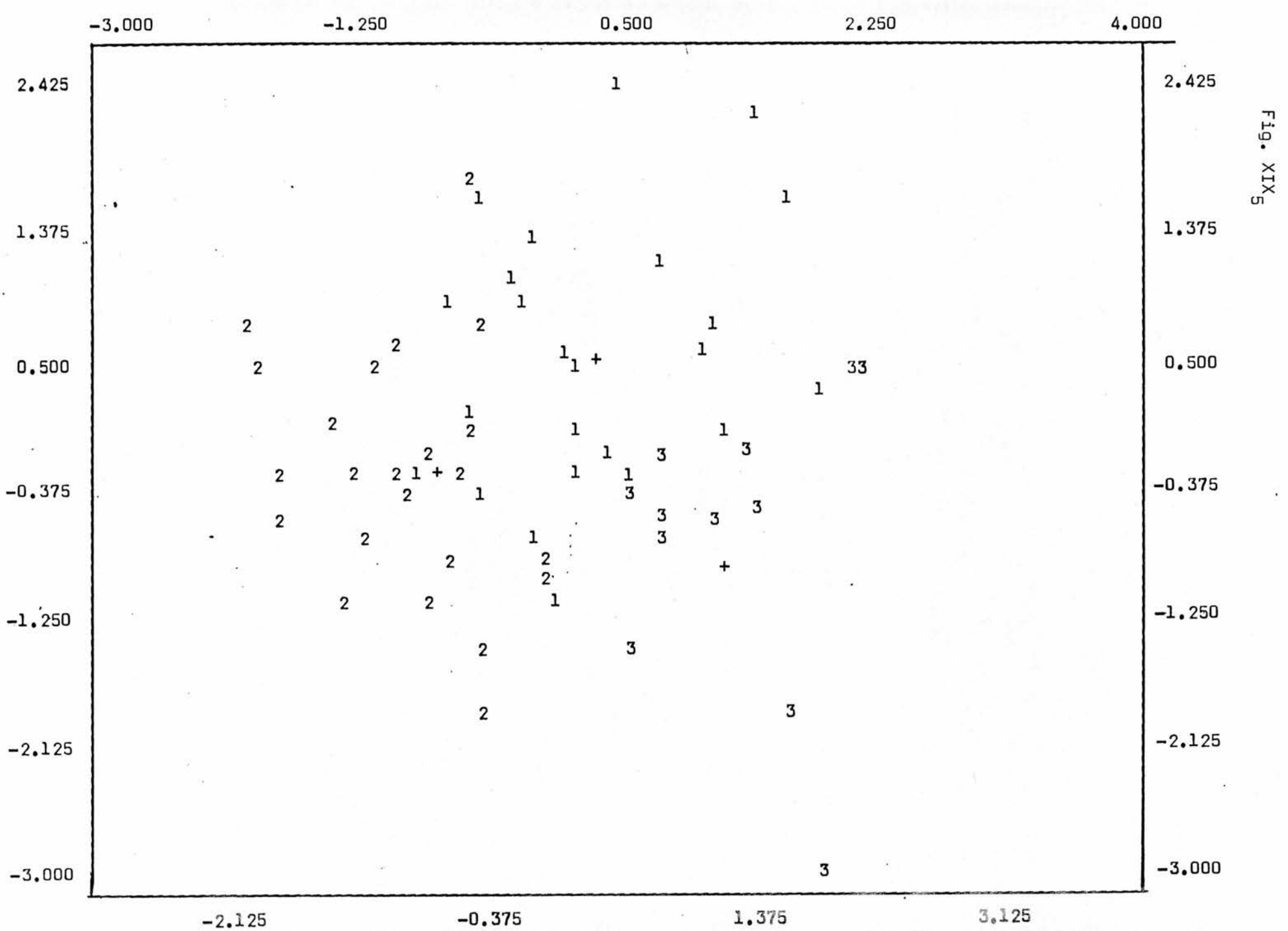

-3.000
-1.250
0.500
2.250
4.000
2.425
1.375
0.500
-0.375
-1.250
-2.125
-3.000
-2.125
-0.375
1.375
3.125

Fig. XIX$_5$

As to the best combination of measurements, the variables which contributed most to function 1 overall were: perceived deviation from normal (WASE = -0.68), PSE total score (PSE = +0.54), pre-morbid functioning (WAS = +0.38), current self-esteem (AM = -0.33), outward irritability (OUT = -0.32), self-reported depression (BDI = -0.30), social class (SOC = -0.29), observed depression (HRS = -0.25) and personal future (FUT = +0.24).

As the mean discriminant scores were well-separated and the overall distribution of scores tended towards distinct separation, these discriminants and their associated psychological meanings may now be applied to the drug and CBT predicted groups.

Table XIX_6 lists the psychological characteristics which further identified the kinds of people who might do best on drugs and CBT.

Table XIX_6 Variables which contributed most towards identifying patients in the predicted groups for drugs and CBT

Drug predicted group	CBT predicted group
1. high PSE total score	1. high perceived deviation from normal
2. good pre-morbid functioning	2. relatively high self-esteem
3. higher social class	3. high outward irritability
4. positive view of personal future	4. high self-reported and observer-rated depression

Thus, these variables, in conjunction with those which predicted a good response to each treatment as gleaned from the multiple regression analysis (see pages in discussion) allow for the development of specific hypotheses concerning the type of people who might respond to combination treatment, cognitive therapy alone, and drugs.